DEPARTMENT OF MEDICINE

# DETECTION OF ASYMPTOMATIC URINARY ABNORMA LITIES IN BUNDELKHAND REGION

## THESIS FOR

## DOCTOR OF MEDICINE
(GENERAL MEDICINE)



## BUNDELKHAND UNIVERSITY, JHANSI (U.P.)

YEAR 2003

ZAHEER HUSAIN

*DEDICATED*

*TO*

*MY TEACHERS*

*&*

*PARENTS*

*WITHOUT WHOM*

*THIS STAGE WAS*

*UNREACHABLE*

# CERTIFICATE

This is to certify that the work entitled "**DETECTION OF ASYMPTOMATIC URINARY ABNORMALITIES IN BUNDELKHAND REGION**" has been carried out by Dr. ZAHEER HUSAIN in the Department of Medicine, M.L.B. Medical college, Jhansi.

He has put in the required stay in the department of medicine as necessitated by university regulations.

Dated: 7/07/03

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This is to certify that the work entitled **"DETECTION OF ASYMPTOMATIC URINARY ABNORMALITIES IN BUNDELKHAND REGION"** which is being submitted as a thesis for M.D. (Medicine) Examination 2003, Bundelkhand University, has been carried out by Dr. ZAHEER HUSAIN under my direct supervision and guidance. The techniques consumed in the preparation of this thesis were under taken by the candidate himself and the observations recorded were checked and verified by me from time to time.

Dated: 07/07/03

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# ACKNOWLEDGEMENT

*It would take pages, to acknowledge everyone who in someway or the other helped me all along my path reaching this goal, but certain individuals need special thanks for their valuable help.*

*First and foremost ,with all my heart I would like to express my deepest gratitude and privilege for having been associated with Dr. P.K.JAIN M.D. MNAMS, Professor , Department of Medicine, M.L.B. Medical College, Jhansi. I feel extremely fortunate and blessed that I got an opportunity to work under his guidance. His constant teaching, supervision, valuable criticism, affection and encouragement has pruned my work time and again in the right direction and has made this difficult task possible for me. His foot prints shall always be a guidance for me in future life and his achievements shall always encourage and help me find success hereafter.*

*I wish to express my humble gratitude and regard to my learned and experienced teacher Professor Dr. R.C. Arora M.D. D.sc., Professor and Head Department of Medicine, M.L.B. Medical College, Jhansi . He has acted as a beacon of light to me through his inspiring guidance , valuable and experienced suggestions and indispensable advices at each step in this study.*

*I am filled with deep sense of obligation towards Dr. N.S.SENGAR M.D.D.M.(Nephrology), Lecturer, Department of Medicine, M.L.B. Medical College, Jhansi. The luminous guidance provided by him in guiding my work time to time has helped me finish this difficult task successfully.*

*I am also greatly thankful to Dr. PRAVEEN KUMAR JAIN M.D. D.M.(Cardiology) and Dr. NAVNEET AGARWAL M.D. Professors Department of Medicine M.L.B Medical College Jhansi for their constant inspiration and guidance throughout.*

*I am thankful to my colleagues and juniors who were a delight to work with and my best friend S.Koshy who was a strong support.*

*Each cell of my body is indebted to my mother Mrs. FARHAT HUSAIN and my father Mr. NAWAB HUSAIN, for their love, affection, care, moral support, blessing and constant encouragement throughout this work . I am also grateful to my brother Mr. SHAHEER HUSAIN and sister Mrs. NIKHAT SULTAN for their constant encouragement.*

*I owe extreme admiration, love, care and thanks to my sweet and loving wife Mrs. SAIRA HUSAIN for her immense love, devotion, concern and help with which she stood by me throughout the testing times. She shall always be my treasure in life.*

*I would like to express my thanks to Mr. Shashank Jain ,chief of Netsol Computers for the pain he took in helping me prepare a presentable and neat work to submit.*

*I shall fail all my thanks if I do not convey all my thanks and respect to all the people who took part in this work and helped it become a complete study which reached conclusion.*

Zaheer Husain

*DATED:* 07/07/03 *(ZAHEER HUSAIN)*

# CONTENTS

# INTRODUCTION

# INTRODUCTION

Near constancy of composition of internal environment of the body, including the volume, tonicity, and compartmental distribution of body fluids and electrolytes is essential to survival.

The kidneys regulate the composition and volume of plasma which in turn determines the composition of volume of intra and extracellular compartments.

Renal diseases may present to physicians in several ways depending upon the nature of the illness and timing of presentation. Some patients present with advanced renal disease, having signs and symptoms of uraemia with unremarkable urinanalysis, while others have urinary abnormalities, but few if any disturbances in renal functions.

## SYMPTOMATIC RENAL DISEASE :

**Can be of 3 Types :**

1. Patients complaining of symptoms or signs which directly or indirectly indicate underlying renal disease.
2. Patients having systemic disease know to be associated with renal involvement.
3. Patients having family history of inherited renal disorders.

Symptomatic renal disease most commonly presents as disorders of micturition, urine volume, urine composition, pain, edema, symptoms of uraemia or symptoms of various disorders involving the kidney secondarily.

**(A) DISORDERS OF MICTURITION:**

Most common disorder of micturition is frequency. Frequency means that the bladder is emptied more often than normal. Frequency may be associated with increased urine volume (polyuria) or normal urine volume. Frequency with normal urine volume can be due to irritation of the bladder by inflammation, stone, tumor or fibrosis. It can also be due to a pelvic mass or gravid uterus. It is important also to determine whether there is normal or decreased volume per void. The former indicates increased urine formation while the latter indicates diminished bladder capacity. Middle aged and older men with prostate enlargement sometimes present with increased urine volume that results from diminished flow rate in nephrons and impaired concentrating. Ability of the kidney due to obstructive back pressure.

**(B) DYSURIA :**

Dysuria is pain, discomfort or burning sensation during micturition, it is usually described by the patient as burning or tingling sensation felt at the urethral meatus or in the suprapubic area during or immediately after micturition. It usually arises as a consequence of bladder, prostatic or urethral inflammation. In younger patients it should be suspected if the child cries during micturition or unexplained fever.

**(C) DISORDERS OF URINE VOLUME:**

Disorders of urine volume can be divided into polyuria (increased volume), oliguria (diminished volume) and anuria (absence of urine).

(a ) Polyuria can be due to :

i. excessive compulsive drinking

ii. increase in tubular solute load (urea in CRF, glucose in Diabetes mellitus or low molecular weight proteins in melanoma)

iii. A diminution in ADH production ( in head trauma, tumor or infections of hypothalamus or pituitary or sleep disorder)

iv. Disordered medullary concentrating ability as a consequence of medullary disease (Analgesic nephropathy, renal papillary necrosis, medullary cystic disease, sickle cell anemia and nephrocalcinosis).

(b) Oliguria describes a reduction in urine volume to less than that required for the excretion of residues from normal daily metabolic function. In adults under extreme conditions, homeostasis can be maintained with urine output of 500ml per day (1.0 ml / kg / hour in young children). Volume less than this is called oliguria. It usually indicates underlying acute renal failure.

(c) Anuria is absence of renal output and indicates obstruction of urinary tract and rarely renal infarction or cortical necrosis.

**(D) DISCOLORATION OF URINE :**

Red / Brown discoloration of urine can be due to certain causes like hematuria, haemoglobinuria, Myoglobinuria, porphyrias, urates, alkaptonuria, drugs and dyes. Blood arising from the glomerulus gives urine a smoky appearance or tea / coca cola appearance. Blood arising from the urethra usually appear at the beginning of urinary stream while that from the bladder or prostate is commonly noticed at the end of micturition.

**( E ) PAIN :**

Pain is an inconsistent symptom of urinary disease but when present is commonly due to obstruction or inflammation. pyelonephritis usually causes pain at renal angle. A perirenal abscess can give symptoms related to diaphragmatic irritation. Glomerular inflammation can be associated with dull lumbar ache. Pain arising from an acute obstruction is usually sudden in onset, severe colicky in nature and radiates from loin to groin.

**( F ) Oedema :**

May arise due to hypoproteinemia which is a consequence of significant proteinuria. The edema is usually most noticed around the eyes in the morning and in the ankles and feet in the evening. Edema may also arise as a consequence of salt and water retention in cases of acute or chronic renal failure.

**( G ) CLINICAL ABNORMALITIES IN URAEMIA :**

Uremia refers to the retention of nitrogenous wastes as renal insufficiency develops and causes multiorgan system derangements which become clinically manifest.

**a. Fluid electrolyte and acid base disorder -**

Can develop and lead to volume expansion/contraction; hypo/ hypernatremia; hypo/hyperkalemia; metabolic acidosis; hypocalcaemia and hyper phosphate.

**b. Endocrine metabolic disturbances -**

Include secondary hyperparathyroidism; vit-D deficiency; Carbohydrate intolerance; hypertriglyceridemia; protein calorie malnutrition; impaired growth; infertility; amenorrhea & sexual dysfunction.

**c. Neuromuscular disturbances-**

Caused due to uremia are fatigue; sleep disorder, headache, impaired mentation, asthenias, seizures, cramps, peripheral neuropathy, myopathy & comes.

**d. Cardiovascular & pulmonary disturbance-**

arterial hypertension; CHF; pulmonary edema; pericarditis; hypertension and arrhythmia.

**e. Dermatologic disturbances-**

Pruritis ; ecchymosed ; hyperpigmentation.

**f. Gastrointestinal disturbances-**

Include anorexia; nausea ; vomiting ; Gastroenteritis ; peptic ulcer ; G.I. bleeding ; peritonitis ; hepatitis.

**g. Hematologic & immunologic disturbances-**

Includes anemia; lymphocytopenia ; bleeding diathesis; infection; splenomegaly.

## *ASYMPTOMATIC RENAL DISEASE*

Is most commonly detected following routine investigations such as urine analysis, blood pressure or blood chemistry analysis after hospitalization for non renal causes, or as part of health screening programs. In a number of patients renal disease is detected during clinical and laboratory test for pregnancy, occupational purposes or health insurance. A considerable number of neonates are diagnosed as having renal disorders because of routine USG screening of mothers during pregnancy. In a small number of cases there is regular screening in view of a known employment and development of renal disease (aniline due workers have a greater incidence of urothelial tumors)

**PERSISTING URINARY ABNORMALITIES WITH NO OR FEW SYMPTOMS.**

The finding of hematuria , proteinuria, bacteriuria , crystalluria and Pyuria in absence of readily identifiable disease are familiar and vexing clinical problems .

**(A) ASYMPTOMATIC HEMATURIA:**

Determining the morphology of erythrocytes in freshly voided urine is useful to separate glomerular form nonglomerular hematuria (fragmented distorted poorly hemoglobinized or dysmorphic RBC with a volume of less than 72 fl are usually glomerular). Glomerular hematuria is frequently accompanied by proteinuria (non nephritic range). Patients with isolated hematuria with minimal urinary symptoms are likely to have no or minimal glomerular changes on electron microcopy and are usually caused by SLE, Wegner's granulomatosis ,Goodpasteur's syndrome , Alport's syndrome , idiopathic hypercalcemia , thin basement membrane , silent Nephrolithiasis or malignancies.

**(B) ASYMPTOMATIC PROTEINURIA:**

Is defined by the pressure of mild glomerular proteinuria (principally albumin) usually less than 2 gm/day with normal urinary sediments in the absence of symptomatic systemic disease. Asymptomatic essential hypertension and mild or latent Diabetic Nephropathy, Idiopathic membranous glomerulonepheritis, Focal glomruloscleresis, IgA nephropathy, and Amyloidosis may all present initially with mild or moderate proteinuria. Other uncommon causes are Postural proteinuria, Over flow proteinuria and Tubular proteinuria.

**(C) ASYMPTOMATIC PYURIA:**

Upto 400,000 cells / hour may be excreted in normal urine, corresponding to 10 WBCs / ml in an unspun urine sample. Many times present of pus cells in the urine can be detected in the absence of clinical symptoms. This is known as asymptomatic pyuria . Common causes of asymptomatic pyuria are infection , Diabetes mellitus, NSAID Nephropathy , Renal tuberculosis , Interstitial nephritis , Nephrolithiasis and prostatic enlargement in males.

**(D) ASYMPTOMATIC GLYCOSURIA:**

Presence of even small amounts of glucose in the urine sample is an abnormality. Asymptomatic glycosuria in individuals is almost always due to diabetes mellitus which has not clinically manifested or detected.

**(E) ASYMPTOMATIC CRYSTALLURIA:**

Except for the cystine crystals and a few others, the majority of crystals found in the urinary sediment are of limited value. It is tempting to associate crystals with a risk of Nephrolithiasis. In a majority of cases, the crystals found in the urine are not present in freshly voided sample . some of the common causes of asymptomatic crystalluria are urolithiasis , infection , primary hyperparathyroidism , excessive bone resorbtion , ethylene glycol toxicity , renal tubular acidosis , chronic diarrhoea and drugs .

# AIMS & OBJECTIVES

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1. To detect the prevalence of asymptomatic urinary abnormalities in Bundelkhand region and to find out the prevalence of diseases that were causing these urinary abnormalities to appear.

2. To compare the results of this study with other studies done in other parts of the country and in different regions of the world. This gave an idea of the changes in prevalence and causes of asymptomatic urinary abnormalities in different age groups in different regions in our country and outside.

3. To show the importance of urinalalysis as a valuable and efficient method of early detection and intervention of urinary abnormalities.

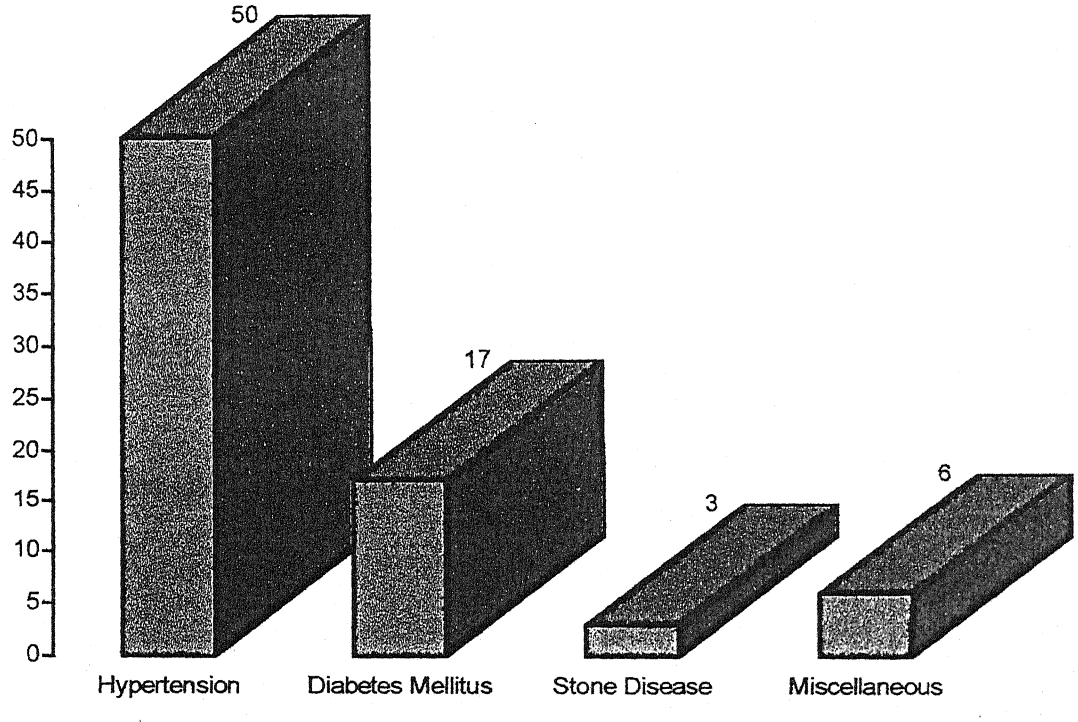
# REVIEW OF LITERATURE

# REVIEW OF LITERATURE

A number of studies have been conducted using urine examination as the tool for detection of asymptomatic urinary abnormalities and surprisingly vast majority of causes have been found out all differing between age, sex, socio economical status and geographic distribution.

Kidney disease detection camp (1994-1996) - A tool for preventive Nephrology conducted by – Vidya N. Acharya, Kumad Mehta et al This study involved 430 subjects seen in 3 Kidney disease detection camps held in the city of Mumbai to cover low socio economical group (LSEG.,nz 244) and mid socio economical group (MSEG;nz 186). The aim was to detect the presence of urinary abnormalities and assess the significance of the same. Subjects attending the camp had their vital data and BP recorded in a specially designated Performa. A fresh sample of urine was obtained from each one of them and examined by using multistix. Those having any clinical or urinary abnormity were reassessed next morning & findings were confirmed. Urinary abnormalities were detected in 42.1% of total subjects (181/430) of which 45.5% were in low-socioeconomic group & 37.6% in mid-socio economic group. In this total of 181 urinary abnormalities 37.8% (111/244) were asymptomatic from LSEG and 47% (70/156) from MSEG. The study found that Asymptomatic urinary abnormalities were associated with hypertension (19.8% in LSEG & 40% in MSEG-P<0.001); Diabetes mellitus (8.1% LSEG & 11.4% in MSEG) and stone disease (1.8%in LSEG & 1.4MSEG).

**Kidney Disease Detection Camp(1994-1996) TYpe of Disease Detected in LSG + MSEG(181)**




In advanced countries like Japan mass screening programmes with compulsory urine screening in children enabled them to detect disease in pre-symptomatic stage successfully. Such studies have been done in adult population too in Japan (all since 1947). During these last 40-50 years, they have been also to detect pre-symptomatic glomerulopathies of various types like IgA nephropathy, membrane proliterative GN etc. and have recommended early treatment for the same. This study her revealed that main causes of urinary abnormalities in our Community has been in association with common maladies like Hypertension (27.6%); Diabetes mellitus (9.4%) which affect the population at large. Simple urine examination on yearly basis would certainly help in early detection of renal involvement and thereby form a valuable tool in the practice of preventive Nephrology.

To define the long-term outcome of patients with minimal urinary abnormalities (defined by the presence of microscopic hematuria with on less than 1 gm/day proteinuria), and normal renal function (defined by a serum creatinine< 1.3 mg/dl), **Shu KH, Ho WL, Lu YS, Cheng CH, Wu MJ, Lian JD Department of Internal Medicine,Taichung Veterans General Hospital, Taiwan** retrospectively studied patients who fulfilled the above criteria and had a kidney biopsy done before the year of 1992 (i.e. at least followed up for 5 years), with a definite pathological diagnosis. A total of 41 cases among 719 cases of primary glomerulonephritis (5.7%) were enrolled into the study. There were 19 males and 22 females with a mean age of 35.4+/-14.7 years at biopsy. The duration of renal disease was 116.0+/-60.5 months and the duration of follow- up post biopsy was 100.2+/-38.1 months. The pathological diagnosis was : IgA nephropathy (21 cases) , focal glomerulosclerosis (9cases), mesangial proliferative glomerulonephritis (8cases) , membranous glomerulonephritis (2cases) and acute glomerulonephritis (1cases) . At the end of follow-up, 8 cases (19.5%) had a certain degree of renal insufficiency including 2(4.9%) in end- stage renal disease. The other cases were either in complete remission (6cases) or stable condition (27cases) with persistent microscopic hematuria and normal renal function. The long-term outcome was not correlated with any of the following parameters: age, sex , disease duration , serum creatinine at presentation, daily protein loss at presentation , degree of glomerular change and degree of tubular atrophy ($P<0.05$) and interstitial fibrosis($P<0.05$).They conclude that a minimal urinary abnormality with normal renal function at presentation does not necessarily imply a favorable long-term outcome in certain patients. Tubular atrophy and interstitial fibrosis but not glomerular change correlates with a worse prognosis. This further emphasizes the importance of renal biopsy in such cases.

**Monhart V, Marek J, Krnch O Ustredni vojenska nemocnice, Praha** studied 103 subjects with asymptomatic isolated haematuria (Persisting for more than 6 months in the absence of proteinuria, bacteriuria, impaired haemocoagulation or urological disease) renal biopsy was preformed. The mean age of the patients was 25.2 years, range 14-58 years. In 94% glomerular changes were detected- most frequently minimal glomerular lesions (67%) and proliferative mesangial glomerulonephritis (15%) . Focal segmental proliferative glomerulonephritis was rare (4%). Immunofluorescent examination revealed IgA nephropathy in 40% (all cases of diffuse and focally segmental proliferative glomerulonephritis and one quarter of minimal glomerular lesions). Changes of tubules and interstitium were recorded in 26%, with the exception of one patient they were always associated with glomerular affection. From the investigation ensues that the predominating cause of isolated asymptomatic haematuria, not clarified by non-invasive examination, is usually not serious and is an affection frequently associated with tubulointerstitial changes. As many as 40% of isolated cases of haematuria may be the manifestation of IgA nephropathy. The deposition of IgA is more frequently associated with a more advanced grade of glomerular affection; Indication of diagnostic renal biopsy in isolated haematuria remains individual.

. **Topham PS, Harper SJ, Furness PN, Harris KP, Walls J, Feehally J Department of Nephrology, Leicester General Hospital , UK,** investigated 165 patients (94 male, 71 female; mean age 37.5 years, range 10-71) referred with isolated microscopic hematuria with normal serum creatinine, no proteinuria, sterile urine and a normal IVU. Renal biopsy abnormalities were found in 77/165 (46.6%). IgA nephropathy (49), global or segmental mesangial proliferative glomerulonephritis without IgA deposits (16) , thin membrane nephropathy (7), vascular changes

suggestive of hypertension (3), interstitial nephritis (1), and membrane nephropathy (1). Only five abnormalities were found on cystourethroscopy (cystitis3, urethral stricture1, and bladder stone 1). Two patients with cystitis also IgA nephropathy. Biopsy abnormalities were commonest under the age of 20(69.2%) , but 40% of biopsies were abnormal even in the seventh decade of life, Because renal biopsy abnormalities are very frequent in patients with isolated haematuria , renal biopsy is indicated in patients over 45 years of age if renal imaging and cystoscopy are normal . In those under 45 years, renal biopsy should replace cystoscopy as the investigation to follow normal renal imaging.

**Hrvacevic R, Dirnitrijevic J, SpasicP, Butorajac J, Jovanovic D** analyzed histopathologic changes in the kidneys of patients with asymptomatic abnormalities of urine analysis if they were correlated with the type of pathologic finding in urine. Retrospective study comprised a total of 76 patients with asymptomatic urine abnormalities. In all three groups of patients, formed upon the type of pathologic finding in urine, were determined heterogeneous histopathologic changes, and different types of glomerulonephritis, respectively. The most frequent histopathologic finding was IgA nephropathy, observed in 16.7% patients with isolated proteinuria, in 50% patients with isolated microscopic hematuria and in 55.9% patients with associated urine abnormalities, In distinction from the other two groups of patients , in the group of patients with isolated proteinuria normal histologic finding was very frequently found (25% patients,) , and in group of patients with asscioted urine abnormalities were observed more severe hestopathologic forms of glomerulonephritis, such as membranoproliferative Glomerulonephritis. It was concluded that different types of glomerulonephritis most frequently caused asymptomatic abnormalities of urine in younger patients. In a

prospective study of idiopathic glomerulonephritis ,**Nieuwhof C, Doorenbs C, Grave W, de Heer F, de Leeuw P, Zeppenfeldt E, van Breda Vriesman PJ, Department of Immunology, University of Limburg ,Maastrict, The Netherlands,** determined the natural history of 49 adult patients ( 12 primary IgA nephropathy, 13 thin GBM nephropathy, 20 normal renal tissue and 4 miscellaneous nephropathies) who presented with idiopathic non-proteinric non-azotemic hematuria of at least six months duration in the absence of hypertension and with a negative urological work- up . The median follow-up was 11 years with a range of 8 to 14 years. At the end of the follow-up, renal function had remained stable in all subsets except for those with miscellaneous disease. Hematuria was still present in all patients with thin GBM nephropathy, in all but two patients with IgA nephropathy who went into immunopathological remission, in three out of four miscellaneous nephropathies, and in seven out of 20 patients with normal renal tissue. Of the latter patients five had a history suggestive of urolithiasis at follow-up, which was in the absence of hypercalciuria and hyperuricosuria. Seven thin GBM patients, five IgA nephropathy patients and three miscellaneous nephropathies developed hypertension; the incidence of hypertension in each subset was significantly higher than in patients with normal renal tissue. This study shows that in young adults with idiopathic chronic non-proteinuric hematuria of four years duration, renal biopsy will give a definite diagnosis in 86% patients , and that those patients with so- called minor glomerular disease are at high risk for hypertension. Those patients with normal renal tissue have a high incidence of urolithiasis and should have a urological follow-up,

In a mass screening programme- **Hisano S, kwano M, Hatae K, Kaku Y, Yamane I, Ueda K, Uragoh K, Honda S Department of**

**pediatrics, Faculty of Medicine, Kyushu University , Fukuoka, Japan--** screened 251 children with isolated microhaematuria . Of these 251 children, 115 were excluded from the study because of microhaematuria secondary to a specific cause. The remaining 136 children were diagnosed as having asymptomatic isolated microhaematuria (ASH). Of these 136 children, 23 had evidence of urinary abnormalities in their family members, Red blood cell casts were evident in 31 children at their initial visit or during the follow-up period. Ten children had one or more episodes of macrohaematuria during the study. Renal biopsy was performed in 19 children because of indications of glomerular disease, and 13 of these 19 children had mild to moderate glomerulonephritis. None of these 136 children developed hypertension or renal impairment after a mean period of 7.4 years ( range6-13 years), Thirty –five children had normal urinary findings within 6 years of their initial visit , and 100 have had persistent microhaematuria with proteinuria greater than 1g/ $m_2$ per day at the end of the study . This study suggests that the prognosis of ASH is good, and that renal biopsy is not indicated for children with ASH.

**Benbassat J, Gergawi M, Offringa M, Drukker A Department of Sociology of Health, Faculty of Health Sciences, Ben- Gurion University , Beer- Sheva , Israel--** reviewed published data on the frequency of underlying disorders in schoolchildren with microscopic or gross isolated haematuria (IH), and evaluated management strategies. They found five reports of microscopic IH in screened asymptomatic schoolchildren, three reports of microscopic IH detected by case-finding, and five surveys of kidney biopsies in referred children with microscopic and gross IH. They listed the reported underlying disorders, and estimated the benefit from their early detecting and treatment. Most children with microscopic IH, whether detected by screening or case-finding, had no

significant underlying disease. Some had disorders that may benefit from early treatment (membranoproliferative glomerulonephritis, obstructive uropathy, urolithiasis), or counselling (hereditary nephropathy, renal cystic disease). The combined prevalence of these five diseases was 0-7.2% in children with microscopic IH detected by screening, and 3.3%- 13.6% in those with microscopic IH detected by case – finding. The combined prevalence of membranoproliferative glomerunephritis and hereditary nephropathy among kidney biopsies was 11.6% -31.6% in children with microscopic IH, and 3.6% -42.1% in children with gross IH, Variable management strategies for schoolchildren with IH result from uncertainly about the frequency of underlying disorders and the efficacy of their early treatment , With no evidence that detecting III leads to prevalence of renal function impairment, screening for IH in symptom less schoolchildren is not warranted. Once detected, however, IH justifies further investigation.

Since 1998, mass urine screening tests have been performed on Korean school children. **Cho BS, Kim SD, Choi YM, Kang HH Department of Pediatrics, College of Medicine, Kyung-Hee University Hospital, 1 Hoegi-dong Dongdaemun-ku, Seoul, Korea** have analyzed those patients who showed abnormal urinary findings in the school screening program. Between January 1998 and January 2000, 452 children with abnormal urinary findings visited the Pediatric Kidney Center, Kyung-Hee University Hospital. Sex, age, 24-h urine creatinine clearance, ultrasonography, Doppler scans and renal biopsies were reviewed retrospectively. Results of initial urinalysis were divided into three groups: solely hematuria group (228 cases, 50.4%), solely proteinuria group (98 cases, 21.7%), and combined hematuria and proteinuria group (79 cases, 17.5%). Among the biopsies cases, the proportions representing renal

parenchyma diseases were as follows: IgA nephropathy 11.3%, mesangial proliferative glomerulonephritis 21.9%, others 3.8%. Among the three groups, the combined hematuria and proteinuria group had more frequent chronic renal disease (57.7%) than the other groups. Chronic renal disease was detected in 36.9% of all visiting subjects. In the school screening program a significant number of patients showed abnormal urinary findings, which were associated with chronic renal diseases especially in the combined hematuria and proteinuria group. In conclusion, mass urine screening tests should be mandatory to detect asymptomatic chronic renal disease in school children.

**Lin CY, Sheng CC, Lin CC, Chen CH, Chou P. Department of Pediatrics, Section of Immunology & Nephrology, Taipei Veterans General Hospital, No. 201, Sec. 2, Shih-Pai Road, Shih-Pai, Taipei, 11217, Taiwan** screened students of public and private elementary and junior high schools in the Province of Taiwan each semester since 1990. About 3 million students were screened each time. The students who had abnormal urine screening results at the first time received a second urine analysis 10 to 15 days later to confirm the abnormal urine analysis. The blood samples of the students with abnormal urine examination were taken and biochemistry examinations including creatinine (Cr) etc. were performed since 1992. All students with abnormal urine screening results were graded by the severity of hematuria and proteinuria, the heavy proteinuria graded as "D". Chronic renal failure (CRF) is defined as impaired renal function with the serum Cr over 1.7 mg/dl. Longitudinal continuous blood and urine examinations were performed each semester for the students of grade "D" and with CRF. CRF was confirmed by either the hospital medical records or telephone visit. The purpose of this study was to delineate the prevalence of heavy proteinuria (grade D) and CRF in

the students of elementary and junior high school in the Taiwan Province from 1992 to 1996. The results revealed the number of urinary screening was 10,288,620. There were 5980 cases with heavy proteinuria with four-year prevalence of 5.81 x 10(-4), 4.83 x 10(-4) for boys; 6.87 x 10(-4) for girls. Girls were affected more often than boys. The peak age of girls was 12 years old and boys were 13 years old. The number of CRF cases was 189 with the four-year prevalence of 1.84 x 10(-5), 2.24 x 10(-5) for boys; 1.41 x 10(-5) for girls. The incidence rate increased after the age of 10; the peak age of boys being 14-year-old and of girls 12-year-old. The exact contributing factors, such as location on islet or lack of pediatric nephrologist, need further study. In conclusion, the four-year prevalence of heavy proteinuria in the students of the elementary and junior high schools in Taiwan was higher in girls than in boys. Glomerular nephritis (GN) is still one of the major causes of urinary abnormalities. The most-important secondary GN was systemic lupus erythematosus (SLE) with lupus nephritis. The percentage of SLE patients among anti-nuclear antibody (ANA) positive was 72%. In contrast, the four-year prevalence of CRF disease was higher in boys with the peak age at 14-year-old. GN is still the major cause of urinary screening abnormality. ANA study is indicated in all Chinese students with abnormal urinary screening.

Clinicohistopathologically, **Takebayashi S, Yanase K. Second Department of Pathology, School of Medicine, Fukuoka University, Japan** observed 109 patients with asymptomatic urinary abnormalities found via the Japanese school medical screening process. Follow-up was for a mean period of 9.3 +/- 4.0 years. More than 80% of the patients had either IgA nephropathy (IgAN, 47 cases, 43.1%), thin membrane disease (TMD; 21 cases, 19.3%) or normal glomerulus (NG; 20 cases, 18.3%). Complete remission appeared in 60.0% of the NG cases, 14.3% of the

TMD cases and in 19.1% of the IgAN cases, and remission was significantly high in the NG group (p less than 0.01). No patient with TMD and NG ever progressed to the extent of pronounced proteinuria or renal failure. One patient deteriorated and required hemodialysis, and 2 patients developed renal insufficiency in IgAN. All of these cases possessed severe glomerular sclerotic change when the initial biopsies were performed. All IgAN cases that went into remission, however, had minor glomerular abnormalities. A positive family history of urinary abnormality was observed in 14.1% of both the IgAN group and the NG group, whereas we observed 71.4% in the TMD group, which was significantly high (p less than 0.01). Other cases included 4 each with non-IgA proliferative glomerulonephritis, focal segmental glomerular sclerosis, membranoproliferative glomerulonephritis and Alport's nephritis.It was concluded that the majority of patients (80.7%) with urinary abnormalities found via the school screening program had IgAN, NG or TMD. 74.5% of the IgAN group and 85.7% of the TMD group had long histories of urinary abnormalities extending into adulthood with no deterioration of the renal function.

**Wei JN, Chuang LM, Lin CC, Chiang CC, Lin RS, Sung FC. Institute of Environmental Health, National Taiwan University College of Public Health, 1 Jen Ai Road, Section 1, 100, Taipei, Taiwan (1993-1999),** did a mass screening programme to describe the gender differences in cases and characteristics of diabetes mellitus (DM) that can be identified from a mass urine screen program for school children in Taiwan.Screening for the childhood asymptomatic proteinuria and glucosuria began in 1992 for school children. Students were instructed to collect mid-stream samples of the first morning urine for glucosuria and proteinuria tests using urine strip devices. Students with positive results for

glucose and/or protein and/or occult blood in the first examination received a second urine test. The third screening test was performed for urine and fasting blood sample for 11-item examinations if the second test was positive. The 1997 criteria of American Diabetes Association were used for defining DM. Approximately 2615000-2932000 students received the preliminary screening each semester. The overall average rates of newly identified diabetes from 1993 to 1999 were 8.3 per 100000 among boys, and 12.0 per 100000 among girls. The average rate of new cases increased significantly from sixth grade for boys and fourth grade for girls, with peak rates of 14.7 per 100000 in eighth grade for boys and 19.0 per 100000 in sixth grades for girls. Similar prevalence trends by sex and grade were observed, higher in girls than in boys. This mass screening data suggest that childhood diabetes of all types in Taiwan is elevated in the age of puberty and higher in girls than in boys.

By governmental mandate, Japanese school children are screened annually for proteinuria, hematuria, and glucosuria to identify children with possible renal disorders. **Pugia MJ, Murakami M, Lott JA, Ohta Y, Kitagawa T, Yamauchi K, Suhara Y, Kasjima J.** added urine dipstick tests for albumin and creatinine to the Japanese screening protocol, and used their dipstick results for blood, glucose and protein. The sulfosalicylic acid precipitation test was used to confirm "trace" positive protein dipsticks. The Japanese and our screening protocol have in common the same data for glucosuria and proteinuria. Their scheme has an algorithm for repeat testing of children with abnormal results, and further testing and medical evaluation for those showing persistently abnormal values. Out of the 23,121 students, we found seven with likely nephritis, one with confirmed nephritis, one with nephrotic syndrome, 170 with persistent unexplained hematuria, 19 with persistent unexplained proteinuria, 14 cases of urinary

tract infection, and 20 cases of likely diabetes mellitus. We conclude that dipstick testing for albumin, protein, creatinine, glucose and occult blood has significant value in a multilevel testing scheme for identifying children with urinary tract abnormalities or diabetes. The assay of albumin increases the sensitivity of the screening, and dividing the albumin by the creatinine concentration reduces the potential errors arising from concentrated or dilute urines.

Beginning in 1974, the Japanese Ministry of Health Welfare directed the screening of schoolchildren for proteinuria. **Pugia MJ, Lott JA, Kajima J, Saambe T, Sasaki M, Kuromoto K, Nakamura R, Fusegawa H, Ohta Y** studied their procedure and methods in 6197 school children and also evaluated a new urine dipstick that measures albumin concentrations down to about 10 mg/l and creatinine down to about 300 mg/l. They used specimens from adult in- and outpatients to test the accuracy of the dipsticks. Based on the quantitative results, they set as cutoffs < 150 mg/l for protein and < 30 mg/l for albumin as the concentrations representing "low risk." The quantitative values were assumed to be correct, and the dipstick results were judged accordingly, i.e., a dipstick protein of > or = "150" mg/l or an albumin of l "30" mg/l indicated increased risk of developing or having a genitourinary disorder. The sensitivity/specificity of the protein dipstick was 95.1%/95.5%, and the same for the albumin dipstick was 83.8%/93.8%. The cut-off for the albumin dipsticks probably should be set somewhat lower to reduce the number of false negatives and increase the sensitivity of the dipstick. When they compared the quantitative albumin to the protein dipsticks with the above cut-offs, they found the sensitivity/specificity to be 79.3%/94.4%, i.e., much like the albumin dipstick results.The many reports on the association of albuminuria and risk of renal disease recommend that

screening should be done for albumin rather than protein. Based on the data from the school children, we estimate that a dipstick albumin of "30" mg/l is borderline increased risk, and that a protein dipstick of "150" mg/l is the same. If they call the dipstick "10" mg/l albumin, "30" mg/l albumin and the "150" mg/l protein results "low risk," then they estimate the prevalence of albuminuria in the school children to be about 2.1% and proteinuria to be about 4.3%. Children with these values should have a quantitative test for albumin and protein. They also tested a dipstick for creatinine and found increasing values with increasing age in both genders; the older boys had significantly higher creatinine values than the older girls and younger boys. For the albumin/creatinine ratio, we found 6028 children with a ratio of > or≥ 30 mg/g indicating low risk and 159 children with a ratio of > or = 30 mg/g indicating increased risk. The ratio may be more useful owing to the likely reduction of the number of false negative and false positives.

Screening urine for microhematuria as an indicator of serious disease is controversial because of the low positive predictive value of such screening and the costs and risks of the associated evaluation. To further evaluate test properties, **Hiatt RA, Ordonez JD. Division of Research, Kaiser Permanente Medical Care Program, Oakland, California 94611** retrospectively examined the outcomes of 20,571 men aged > or = 35 years and women aged > or = 55 years who voluntarily had a Personal Health Appraisal in 1980 as members of a large prepaid health plan. Hematuria was detected by dipstick in 876 cases (4.3%); 278 were excluded because of evidence of previous urological disease which could cause hematuria. Review of the medical records of 598 patients with asymptomatic microhematuria as shown by a positive dipstick result indicated that 99% had a follow-up evaluation within 3 months of positive

test results for hematuria and had various levels of urological evaluation thereafter. However, urological cancers (2 prostate, 1 bladder) developed in only 3 patients within the next 3 years. On the basis of San Francisco-Oakland Surveillance, Epidemiology, and End Results program data, rates of urological cancer were evaluated among patients whose test results were negative for hematuria, and these cancer rates were found to be almost the same as the rate among patients with asymptomatic microhematuria. Sensitivity of a single dipstick urinalysis result using microhematuria to indicate urological cancer within 3 years was 2.9%; specificity was 96.7%; and positive predictive value was 0.5%. Multivariate analysis which adjusted for age, gender, and race showed that the relative risk of 2.1 (95% confidence interval, 0.7-6.6) for urological cancer was not significantly elevated among patients with asymptomatic microhematuria compared with patients who had negative test results. These findings based on a single test are consistent with the current lack of recommendations for screening for microhematuria among asymptomatic adults.

The prevalence and incidence of renal diseases in developing countries are not known. This lack of knowledge is an obstacle to the adoption of preventive measures which may be of great value in a social and economic environment where treatment options for end-stage renal failure are simply not available to the vast majority of the population. Urinalysis, a simple and inexpensive test, remains a cornerstone in the evaluation of the kidney and may also be easily employed in mass screening for renal abnormalities in a developing country. **Plata R, Silva C, Yahuita J, Perez L, Schieppati A, Remuzzi G. Mario Negri per L'America Latina, Renal Diseases Project, Department of Nephrology and Dialisis Hospital Juan XXIII, La Paz, Bolivia.** Conducted an

educational campaign on renal diseases in three selected areas of Bolivia. Urine samples were collected and sent to one of 21 participating clinical centers. Fresh urine specimens were screened using a dipstick for chemical analysis and by microscopic urinalysis after centrifugation. In those patients in whom urinary abnormalities were found, further investigations were carried out in order to define the diagnosis; these patients were enrolled in a 3-year follow-up program. Apparently healthy subjects (n = 14,082) were referred to the First Clinical and Epidemiological Program of Renal Diseases from rural and metropolitan areas in Bolivia. Urinary abnormalities were detected in 4261 subjects at first screening. The most common form of urinary abnormality was hematuria, which was found in 2010 (47% of positively screened subjects). Other renal abnormalities were leukocyturia (41%) and proteinuria (11%). Confirmatory tests and further clinical studies were then carried out in 1019 people. On a second screening 35% of the subjects had no urinary abnormalities; in the remaining people the following diagnosis were made: asymptomatic urinary-tract infection (48.4%), isolated benign hematuria (43.9%), chronic renal failure (1.6%), renal tuberculosis (1.6%). Other diagnosis were: renal stones 1.3%, diabetic nephropathy 1% and polycystic kidney diseases 1.9%. CONCLUSIONS: This study helped define for the first time the frequency of asymptomatic renal diseases in Bolivia. It shows that it is possible to screen a large population of patients at relatively low cost, providing the framework for further action that may help in the prevention and timely diagnosis of renal diseases.

A possible method of improving the prognosis of bladder cancer may be the widespread introduction of screening. **Britton JP, Dowell AC, Whelan P, Harris CM. Department of Urology, St. Jame's University Hospital, Leeds, United Kingdom,** investigated the ability of urine

dipsticks to detect early bladder cancer in a group of men in the community. In 2,356 men more than 60 years old the urine was tested with a dipstick for the presence of blood. The subjects then tested their own urine on 10 subsequent occasions. Of the men 474 (20%) had dipstick hematuria and 319 agreed to undergo urological investigation. An asymptomatic bladder tumor was found in 17 men, associated in 10 with abnormal urine cytological findings. Urine dipsticks for the detection of red cells provided an inexpensive, simple and acceptable screening test for bladder cancer. However, introduction of generalized population screening by this method would produce large numbers requiring investigation. Combining urine cytology with dipstick hematuria results may provide a realistic alternative and further evaluation of the effectiveness of screening for bladder cancer in the community is required.

Microalbuminuria (MA) is associated with adverse health outcomes in diabetic and hypertensive adults. The prevalence and clinical significance of MA in nondiabetic populations is less clear. **Jones CA, Francis ME, Eberhardt MS, Chavers B, Coresh J, Engelgau M, Kusek JW, Byrd-Holt D, Narayan KM, Herman WH, Jones CP, Salive M, Agodoa LY. Division of Genetics and Epidemiology, Joslin Diabetes Center, Boston, MA, 02215, USA.** camille.jones@joslin.harvard.ed did a study to generate national estimates of the prevalence of MA in the US population. Untimed urinary albumin concentrations (UACs) and creatinine concentrations were evaluated in a nationally representative sample of 22,244 participants aged 6 years and older. Persons with hematuria and menstruating or pregnant women were excluded from analysis. The percent prevalence of clinical proteinuria (UAC > or = 300 mg/L) was similar for males and females. However, the prevalence of MA (urinary albumin-creatinine ratio [ACR], 30 to 299 mg/g) was significantly lower in

males (6.1%) compared with females (9.7%). MA prevalence was greater in children than young adults and increased continuously starting at 40 years of age. MA prevalence was greater in non-Hispanic blacks and Mexican Americans aged 40 to 79 years compared with similar-aged non-Hispanic whites. MA prevalence was 28.8% in persons with previously diagnosed diabetes, 16.0% in those with hypertension, and 5.1% in those without diabetes, hypertension, cardiovascular disease, or elevated serum creatinine levels. In adults aged 40+ years, after excluding persons with clinical proteinuria, albuminuria (defined as ACR > or = 30 mg/g) was independently associated with older age, non-Hispanic black and Mexican American ethnicity, diabetes, hypertension, and elevated serum creatinine concentration. MA is common, even among persons without diabetes or hypertension. Age, sex, race/ethnicity, and concomitant disease contribute to the variability of MA prevalence estimates. 

To elucidate prognosis and prevalence of chronic renal diseases among proteinuric and/or hematuric subjects found in mass screening, a long-term follow-up study (6.35 years, range 1.03-14.6 years) was conducted on Japanese working men by **Yamagata K, Takahashi H, Tomida C, Yamagata Y, Koyama A. Institute of Clinical Medicine, University of Tsukuba, Japan. k-yamaga@md.tsukuba.ac.jp** A total of 772 subjects selected from 50,501 Japanese men aged 15-62 years were found to have asymptomatic hematuria (n = 404), concomitant hematuria and proteinuria (n = 155), and proteinuria (n = 213) during their annual urine examination and five consecutive urinalyses. Hematuria patients showed significant improvements in urinary abnormalities as compared with both hematuria/proteinuria and proteinuria patients. Both hematuria/proteinuria patients with normotension and

hematuria/proteinuria patients aged under 40 years showed significant improvements. During the follow-up period, 9.5% of the hematuria patients became hematuric/proteinuric. Hematuria/proteinuria patients had the highest risk of developing renal insufficiency. The presence of hypertension at detection of urinary abnormalities did not affect the renal function; however, if proteinuria appeared after the age of 40 years, these patients had a higher risk of developing renal insufficiency. The incidence of IgA nephropathy in the present subjects was as high as 143 cases per 1 million per year. Detailed follow-up and definitive diagnosis of asymptomatic urinary abnormalities may raise the prevalence of IgA nephropathy worldwide. Copyright 2002 S. Karger AG, Basel.

The incidence of asymptomatic bacteriuria is reported as 2-14% during pregnancy. Fetal and maternal complications like acute pyelonephritis, hypertension, anemia, preterm labor, low-birth-weight infants and intrauterine growth retardation can be expected. The purpose of this study was to determine the incidence of asymptomatic bacteriuria during pregnancy and its relation to pregnancy complications. The study involved 270 pregnant women up to 32 gestational weeks during a 9-month period. At the initial visit, they were screened with urine culture in order to detect asymptomatic bacteriuria. A control group was formed in a retrospective manner from the first day of the study with 186 pregnant women who delivered in our clinic and who were not screened for asymptomatic bacteriuria. The incidence of asymptomatic bacteriuria was 9.31%. Escherichia coli accounted for 79%, which was the most frequent of the isolates. We observed recurrence and had to apply treatment again to 21.7% of the women. The sensitivity, specificity, positive predictive and negative predictive values of leucocyturia as a screening test for asymptomatic bacteriuria were 91.3%, 83.6%, 45.6% and 98.5%,

respectively. We diagnosed preterm labor in six of 23 (26%) with asymptomatic bacteriuria and 16 in 163 (9.3%) women in the urine culture negative group. The ratio acute pyelonephritis in the group which was routinely screened and treated for asymtomatic bacteriuria was 0.5% while the prevalence was 2.1% in the nonscreened group. Considering the relatively high incidence of asymptomatic bacteriuria during pregnancy and the relevant complications, we propose to screen and treat asymptomatic bacteriuria routinely in all pregnant women.

# MATERIAL & METHODS

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To accomplish detection of asymptomatic urinary abnormalities in Bundelkhand region, it was necessary to screen the population in an effective and cheap way. Hospital was a biasing factor as most population of different regions draining into the hospital was symptomatic and suffering from variety of disease that may primarily or secondarily involved the renal system.

The best way was to conduct renal disease detection camps for the mass population in the study area with the help of health clubs and health promoting missionaries and projects.

The second problem was cost and effectiveness of the method used for screening. Eventually it was realized that uroscopy (cytobiochemical examination of urine) was a cheap and fruitful examination to screen for asymptomatic renal disease load in the population.

While screening the mass, a working format was decided for each individual attending the renal camps that included Name, Age, Sex, and Blood pressure, symptoms at presentation, urine examination, blood glucose examination and fundus examination. Individuals who had detectable urinary abnormalities were called the next day for repeat and further evaluation of the abnormalities and for reaching at a clinical diagnosis as the cause of this urinary abnormality. Of this mass of population screened a significant fraction was found to have detectable urinary abnormality and many of such patients were without clinical symptoms.

Taking the study conducted by Dr.V.N. Acharya in Bombay city as prototype, this study was conducted into 2 parts. First part comprised on initial assessment of urine (macroscopic and microscopic), blood pressure, and clinical symptoms of presenting patients and the second part was a follow up of cases that were detected to have urinary abnormality, so as to confirm the persistence of the urinary abnormality detected initially by repeat urine examination and work up and reach the cause of that persistent urinary abnormality.

For each part of this study simple working proforma were laid down and each patient that attended our renal camps was assessed according to this preplanned proforma.

## *WORKING PROFORMA 1 (Renal camps):*

Included the following data for all group of people attending the renal camps :-

1. **Name :**

2. **Age :**

3. **Sex :**

4. **Blood pressure:** was recorded using mercury manometer with a 12 cm broad cuff. For children below 12 years an 8 cm duff was used. For correct recording the deflation rate was usually kept at 2mm per second.

5. **Clinical symptoms:** Any symptoms complained by the patient that appeared to be urological, were recorded. Patients with symptoms other than renal were considered to be asymptomatic.

## 6. Fresh Urine examination(Routine and Microcopy) :

### COLLECTION:

There are 3 ways to obtain a urine specimen – spontaneous voiding, urethral catheterization, and suprapublic bladder puncture. Spontaneous voiding is the simplest and best method, if specimen is collected appropriately. A clean catch urine sample was obtained. In males, foreskin was retracted and glans penis cleansed. Similarly, in females, the labia were separated and area of labia and urethral meatus cleansed. Then midstream urine was collected .The present study used the spontaneous voiding method .

### MACROSCOPIC EXAMINATION:

**Urine sugar:-**

In this study urine sugar was tested by benedict's method

BENEDICT'S TEST: - This test is based on the ability of sugar in urine to reduce cupric ions to cuprous ions. Though the test is not specific but it is better and more specific than the fehling's solution test.

Benedict's qualitative reagent – was prepared by dissolving 173 g of sodium citrate and 100 g of anhydrous sodium carbonate in about 600 ml warm water. This was filtered into a one liter volumetric flask.

Then 17.3 g of copper sulphate ($CuSO4.\ 5H_2O$) in about 100 ml water in a beaker and added slowly to the above mixture with constant shaking .This final mixture was made 1 litre by adding water.

Procedure: - 5 ml of Benedict's reagent was taken in tube and to it 0.4 ml (8 drops) of urine was added. The tube was heated directly on the flame till

the solution boiled. This was then cooled and examined for change of color.

| Observation | Clinical record | Approx.glucose concentration ( gram/100 ml urine) |
|---|---|---|
| Blue | 0 | Nil |
| Green(no precipitate ) | ± | Traces |
| Green(with precipitate) | + | 0.5 |
| Brown | + + | 1.0 |
| Orange | + + + | 1.5 |
| Red | + + + + | 2.0 and over |

Note: - In addition to glucose the reduction of Benedicts solution may be caused by the following (false positive): (i) other sugars including lactate, fructose and pentoses. Lactose is the commonest, particularly during late pregnancy or lactation. (ii) Normal urine constituents, particularly uric acid, creatinine and ascorbic acid. Reduction is slight and only occurs with concentrated urines. (iii) The end products of drugs, commonly aspirin and salicylate (which are excreted as glucuronides and salicyluric acid).

**Urine protein**: – The presence of protein in urine is called proteinuria. Albumin is the main constituent, though higher molecular weight globulins also appear in the urine. Various simple methods for testing urine protein are available.

HEAT PRECIPITATION TEST- In this test the adjustment of urine pH to about 5 is necessary because the proteins only coagulate when they are heated at a pH near their isoelectric point (pH 5). False negative results may otherwise be obtained if the urine is alkaline or more acidic. Furthermore, by adjusting the pH before heating, the difficulties caused by the precipitation of phosphates can be avoided.

Reagents used :-

Acetic acid 33% solution.

Procedure – Take a test tube almost full of clear urine and test the pH with litimus paper or narrow range pH paper and adjust by drop wise addition of 33 percent acetic acid until it is slightly acidic (about pH5) . Heat the top few centimeters of the urine column to boiling and note any turbidity by comparison with the unheated part of the liquid. Appearance of turbidity or precipitate confirms the presence of protein.

## MICROSCOPIC EXAMINATION:

Preparation of urine sediment is the first step in microscopic analysis. The importance of standardization of technique and quality accuracy cannot be over stressed to ensure accurate and reproducible analysis. Important steps include centrifugation, resuspension of sediment, slide preparation, and microscopic examination. In brief 10 ml of urine was centrifuged at approximately 2,000rpm (1,000-2,500rpm) in a centrifuge machine for 5 minutes. The supernatant 9ml was discarded and sediment was resuspended in 1ml. A drop of this was pipette onto a slide and a cover slip placed. The slide was then examined without staining. The slid was examined both under light and high power field in the microscop the following:

a). **Pus cells** : A value of more than 6 pus cells / HPF was c significant in this study.

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a). **Pus cells** : A value of more than 6 pus cells / HPF was c significant in this study.

b). **RBCs**: A value of more than 5 RBCs / HPF was considered significant in this study.

c). **Crystals**: presence of any number or variety of crystals was considered as urinary abnormality in this study

## 6. Blood glucose:

Depending upon clinical symptoms and urine examination results, a blood glucose examination was done where needed. Conditions like, presence of sugar or protein, or pus cells in urine, presence of hypertensive and symptom like obesity, polyuria, nocturnal, polydipsia all demanded a blood glucose examination and so it was conducted in all such cases.

**7. Fundoscopy:** Fundus was examined in all cases having diabetes mellitus or hypertension to check diabetic retinopathy or vessel changes suggestive of hypertensive.

*WORKING PROFORMA 2 (follow up):*

This included at our hospital the following examination and information.

**1. Name:**

**2. Age:**

**3. Sex:**

**4. Repeat Blood pressure:**

**5. Clinical Symptoms:** Presence of clinical symptoms suggestive of renal disease were asked for and signs for renal disease looked for.

**6. Repeat urine(routine/microscopy) :** Same method was used as used in the working proforma 1.

**7. Serum creatinine :**

Principle : Creatinine is treated with picric acid in alkaline medium , a red colour develops which is measured colorimetrically. The reaction is not specific but at least over 85 percent colour is due to creatinine

Reagents used :-

-Sodium tungstate 10 %

-Sulphuric acid (2/3 N)

-Sodium hydroxide, 10 percent

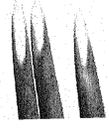
-Saturated picric acid solution

-Stock creatinine standard – Dissolve 100mg of pure dry creatinine in 100mg of 0.1 N-HCl

-Working creatinine standard-Dilute 1.0 ml stock solution to 10 ml with water.

-Alkaline picrate solution- Prepare just before use a mixture of 10 ml saturated picric acid and 2 ml sodium hydroxide .

Procedure:-



Test – in a centrifuge tube 1.0 ml serum was taken. 4 ml water and 0.5 ml sodium tungstate and 0.5 ml sulphuric acid was added to the serum. Solution was mixed by inversion and centrifuged after some time. 3 ml supernatant was taken in another tube.

Standard – 3 ml working standard

Blank – 3 ml water.

1.5 ml alkaline picrate solution was added to each tube. Mixed well and allowed to stand for 10 minutes. The absorbance using green filter (520 nm) against the blank was measured. Calculation :

$$\text{Serum creatinine} = \frac{T}{S} \times 6 \text{ (mg/100ml)}$$

The normal range of serum creatinine is 0.1 to 1.2 mg/100ml.

Increased values are usually found in advanced cases of renal disease. With severe renal failure it may rise to over 10 mg/100ml.

**8. Repeat Blood Glucose (Fasting):** Repeat blood glucose was done. This value was fasting as all cases called for follow up were told to stay 10 hour empty stomach before coming for follow up.

**9. Urine Culture:** Was done in all patients found to have pus cells in their urine. A clean catch sample was collected in a test tube and a loopful was inoculated on 2 types of media for 24 hours. The 2 media used were MacConkey's agar and Blood agar . Growth was observed for and interpreted as follows: More than $10^5$ organisms per ml indicated definite infection from that species, between $10^2$ and $10^5$ organisms indicated possible infection and less than $10^2$ organisms from a single strain excluded infection .

**10. Renal biopsy:** Biopsy was not done in all patients found to have urinary abnormalities. It was only done in cases where the diagnosis was not clear. It was done in the morning after an overnight fasting. An i.v. access was established and maintained for the next 24 hours. Prior to the biopsy the biopsy sight was localized by USG. The Patient was made to lie in a proned position and skin cleaned, draped and infiltrated with local anesthetic agent. A seven inches needle (mostly lumbar puncture needle) was used to explore the kidney (movement of the needle was looked for during respiration).the biopsy needle used by in this study was a true cut needle (11.4cm). After adjusting the length of the true cut needle the biopsy was done. Adequacy of the sample was checked. If inadequate sample, the procedure was repeated. After taking adequate sample, it was transferred into a formalin (10 %) solution in a test tube and send for histopathology and immunoflourescent study. Results were usually obtained within 2-3 days. Care was taken in regards to the patient for the next 24 hours and features like post biopsy hematuria and hypertension and hypotension were specially monitored for in these 24 hours

**11. Final diagnosis:** Using above mentioned methods and procedures cases detected to have asymptomatic urinary abnormalities were diagnosed of their underlying disease.

# OBSERVATION

# OBSERVATION

There were 279 patients seen in various renal camps, held in the region of Bundelkhand and their distribution was as follows :-

Table No I ( CONSTITUENT POPULATION IN STUDY)

| Age Range(years) | Males | Females | Total |
|---|---|---|---|
| 0-12 Yrs | 09 | 16 | 25 |
| 12-20 Yrs | 13 | 14 | 27 |
| 21-40Yrs | 81 | 49 | 130 |
| 41-60Yrs | 53 | 24 | 77 |
| Above 60 yr | 16 | 04 | 20 |
| **Total** | 172 | 107 | 279 |

As shown in table No. 1 a total of 279 people were included in this study. Out of these 279 people the male population was 172 patients making 61.65% of study population and there were 107 females making 38.35% of the study population.

Population upto 12 year of age (paediatric group) included 25 people that is 8.96% of the total, in which there were 9 males (3.22%) and 16 females (5.73%).

The male is to female ratio as a whole in the study population was 1.6:1.

Maximum number of people attending the Renal camp and turning up for re-evaluation were of the age group 21-40 year and minimum number belonged to the age group above 60 years.

Table II (A)

URINARY ABNORMALITY DETECTED IN THE STUDY POPULATION

| Age group (years) | No urinary abnormality | Urinary abnormality detected | Total | % with urinary abnormality |
|---|---|---|---|---|
| 0-12 | 19 | 06 | 25 | 24% |
| 13-20 | 23 | 04 | 27 | 14.81% |
| 21-40 | 104 | 26 | 130 | 20% |
| 41-60 | 53 | 24 | 77 | 31.16% |
| 61 and above | 12 | 08 | 20 | 40.00% |
| **Total** | 211 | 68 | 279 | 24.37% |

As a whole it was found, that there were 68 patients out of 279 screened who has detectable urinary abnormalities both during initial screening and follow up study. This made up about 24.37% of the total. Considering different age groups it was 24% in age group 0-12 ; 14.8% in age group 13-20 ; 20% in Age group 21-40% ; 31.16% in age group 41-60 and 40% in age group above 61 years and above. The maximum percentage of urinary abnormality detected was in the age group 61 years and above (40%) and the minimum percentage was for the age group 13-20 years (14.81%).

TABLE IIB

PATIENTS HAVING ASYMPTOMATIC URINARY ABNORMALITIES

| Age group (years) | Symptomatic Or no urinary abnormality | Asymptomatic Urinary abnormality | Total | % of Asymptomatic urinary abnormalities |
|---|---|---|---|---|
| 0-12 | 21 | 04 | 25 | 16% |
| 13-20 | 24 | 03 | 27 | 11.11% |
| 21-40 | 108 | 22 | 130 | 16.92% |
| 41-60 | 62 | 15 | 77 | 19.48% |
| ≥ 61 | 14 | 06 | 20 | 30.00% |
| **Total** | 229 | 50 | 279 | 17.9% |

As seen in table II B, out of 279 patients screened, 50 patients were detected to have asymptomatic urinary abnormalities, as compared to 68 patients detected to have urinary abnormalities (symptomatic or asymptomatic). This was 17.9% of the total population screened.

For different age groups, the distribution of asymptomatic urinary abnormalities varied, being maximum in the age group above 61 years (30.00%) and minimum in the age group 12 – 20 years(11.11%) , the percentage of other age groups lying between these two as shown in the above table .

Table III.

SEX DISTRIBUTION OF ASYMPTOMATIC URINARY ABNORMALITIES DETECTED

| Age Group | Male ( no. of cases ) | | | Female ( no. of cases ) | | |
|---|---|---|---|---|---|---|
| | No Urinary Abnormalities | Urinary Abnormalities | % | No Urinary Abnormalities | Urinary Abnormalities | % |
| 0-12 | 08 | 01 | 12.50% | 13 | 03 | 18.75% |
| 13-20 | 12 | 01 | 7.60% | 12 | 02 | 14.28% |
| 21-40 | 67 | 14 | 17.28% | 41 | 08 | 16.32% |
| 41-60 | 45 | 08 | 15.09% | 17 | 07 | 29.16% |
| 61-above | 11 | 05 | 31.25% | 03 | 01 | 25% |
| **Total** | 143 | 29 | 20.27% | 86 | 21 | 24.41% |

The male to female ratio for total and asymptomatic urinary abnormalities was as shown in Table III

(1) For the age group 0-12 years :- Asymptomatic Urinary abnormalities were detected in 12.5% of males and 18.75% of females.

(2) For Age group 13-20 year:-Asymptomatic Urinary abnormalities were detected in 7.6% of males and 14.28% of females.

(3) In Age group 21-40 years:- Asymptomatic Urinary abnormalities were detected in 17.28% of males and 6.32% of females.

(4) In Age group 41-60 years:-Asymptomatic Urinary abnormalities were detected in 15.09% of males and 29.16% of females.

(5) In Age above 60 years:-`Asymptomatic urinary abnormality was seen in 31.25% of males and 25% of females.

# DIFFERENT URINARY ABNORMALITIES DETECTED

## (A) PROTIENURIA

### Prevalence of Asymptomatic Proteinuria

TABLE NO. VI

PATIENTS HAVING ASYMPTOMATIC PROTEINURIA AND ISOLATED PROTEINURIA

| Age group (yr) | Total Asymptomatic Urinary Abnormalities Detected | asymptomatic Proteinuria ( %) | Isolated Proteinuria (%) |
|---|---|---|---|
| 0-12 | 04 | 02(50%) | 01(25%) |
| 13-20 | 03 | 00(0%) | 00(0%) |
| 21-40 | 22 | 06(27%) | 06(27.27%) |
| 41-60 | 15 | 08(27.27% | 06(40% |
| 61& above | 06 | 03(50% | 01(16.66% |
| **Total** | 50 | 19(38%) | 14(28%) |

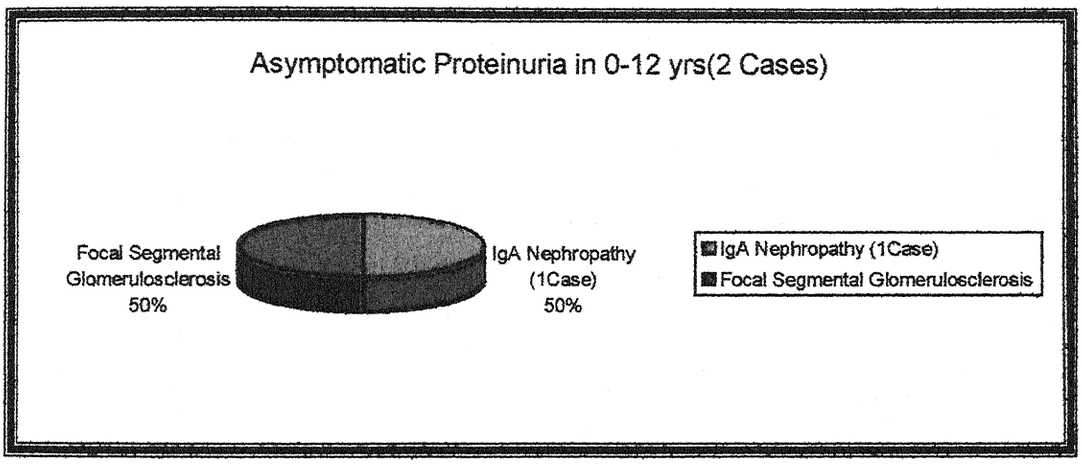
Of the various urinary abnormalities detected, proteinuria was a commonly detected abnormality. It occurred either as isolated proteinuria or along with other abnormalities like pyuria, RBCs, or oxalate crystal etc.

In the age group of 0-12 yrs asymptomatic proteinuria constituted 50%, of which isolated proteinuria constituted 25% of all asymptomatic urinary abnormalities for this age group. Proteinuria was not detected in the age group 13-20 years. Again in age group 21-40yrs proteinuria constituted 27.27% of asymptomatic urinary abnormalities for that age group out of which all were isolated proteinuria. For age group 41-60yrs proteinuria constituted 53.33% of which isolated proteinuria was 40% of all asymptomatic urinary abnormalities for the age group. In 61 years and above, proteinuria was 30% of which isolated proteinuria was 16.66% of all sympatric urinary abnormalities for that age group. Out of 50 asymptomatic

urinary abnormalities 19 were proteinuria of which 14 were isolated proteinuria. Thus Proteinuria constituted 38% of all asymptomatic urinary abnormalities and isolated proteinuria constituted 28% of all asymptomatic urinary abnormalities.Thus out of the study population of 279 people screened asymptomatic proteinuria was found in 6.81% of people (19 cases) and isolated proteinuria was found in 5.02% people (14 cases).

**Causes of Asymptomatic Proteinuria**

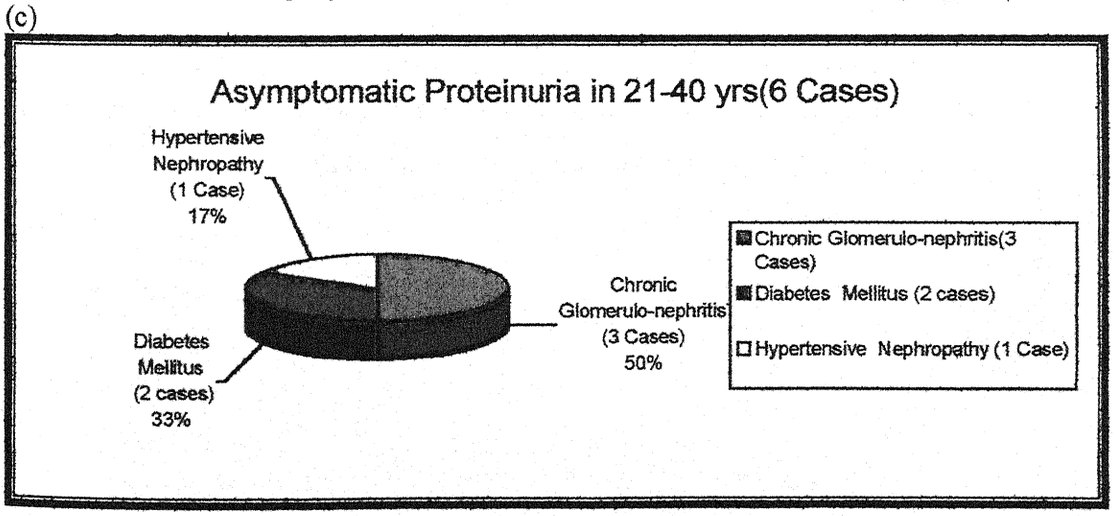
(a)



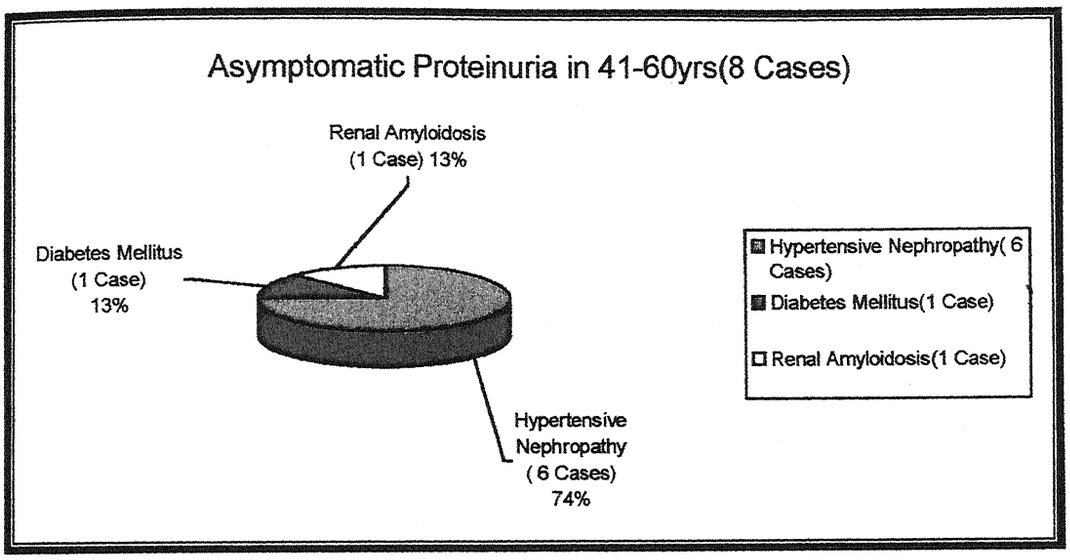

(b)

Asymptomatic proteinuria in 13-20 yrs ( 0 cases )
No cases of Asymptomatic Proteinuria were seen in this age group

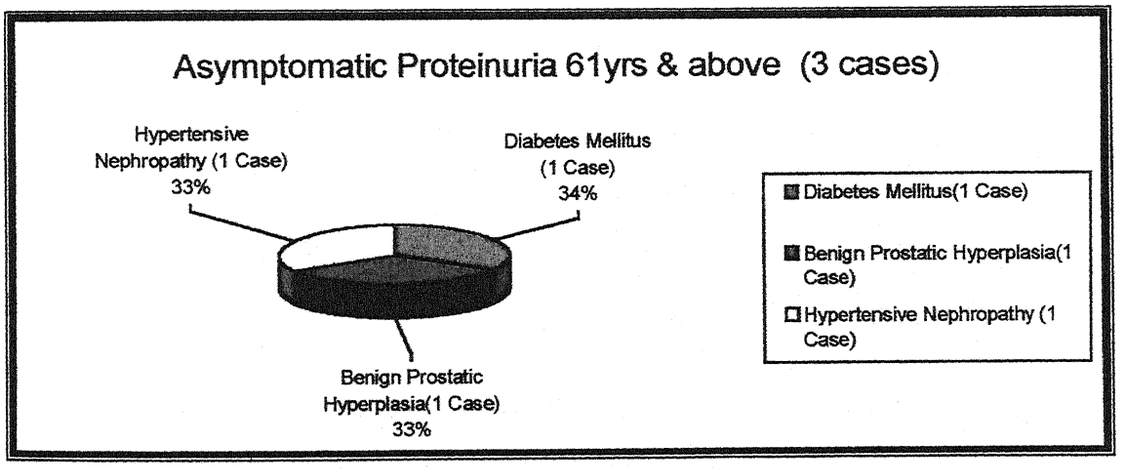
(c)

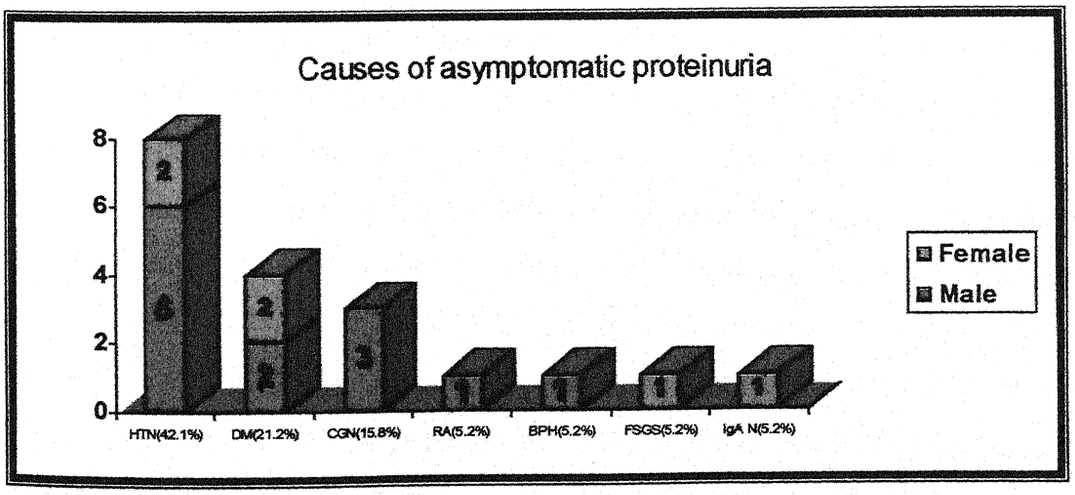


(d)


Asymptomatic Proteinuria in 41-60yrs(8 Cases)
Renal Amyloidosis
(1 Case) 13%
Diabetes Mellitus
(1 Case)
13%
Hypertensive
Nephropathy
( 6 Cases)
74%
Hypertensive Nephropathy( 6 Cases)
Diabetes Mellitus(1 Case)
Renal Amyloidosis(1 Case)

(e)


Asymptomatic Proteinuria 61yrs & above (3 cases)
Hypertensive
Nephropathy (1 Case)
33%
Diabetes Mellitus
(1 Case)
34%
Benign Prostatic
Hyperplasia(1 Case)
33%
Diabetes Mellitus(1 Case)
Benign Prostatic Hyperplasia(1 Case)
Hypertensive Nephropathy (1 Case)


Causes of asymptomatic proteinuria
8
6
4
2
0
HTN(42.1%)
DM(21.2%)
CGN(15.8%)
RA(5.2%)
BPH(5.2%)
FSGS(5.2%)
IgA N(5.2%)
Female
Male

As shown in the above table it was seen that the major causes of asymptomatic proteinuria was Hypertensive nephropathy ( 42.1%) , Diabetes mellitus (21.2%) , and Chronic glomerulonephritis ( 15.8%) , other causes being Renal amyloidosis , Benign prostatic hyperplasia , Focal segmental glomerulosclerosis , and IgA Nephropathy .

**(B) ASYMPTOMATIC PYURIA**

**Prevalence of Asymptomatic Pyuria**

Table No. V

PATIENTS DETECTED HAVING ASYMPTOMATIC PYURIA

| Age Group | Total Asympt. Urinary Abnormalities | Asymptomatic Pyuria with other abnormalities | Asymptomatic Isolated Pyuria |
|---|---|---|---|
| 0-12 | 04 | 01(25%) | 01(25%) |
| 13-20 | 03 | 03(100%) | 03(100%) |
| 21-40 | 22 | 11(50%) | 07(31.8%) |
| 41-60 | 15 | 07(46.66%) | 05(33.33%) |
| 61& above | 06 | 04(66.66%) | 01(16.66%) |
| **Total** | 50 | 26(52.00%) | 17(34%) |

Another urinary abnormality commonly encountered in this study was pyuria. Pyuria was detected very commonly and more so in young females. The male to female ratio in all asymptotic cases of pyruia detected was 14:12. For age group 0-12 years 25% of all asymptomatic urinary abnormalities was pyuria of which all was isolated pyuria. For age group 13-20, the prevalence was 100% of all asymptomatic abnormalities (all asymptomatic urinary abnormalities were isolated pyuria). For age groups 21-40 years prevalence of pyuria was 50% of which isolated pyuria was 31.8% of all asymptomatic urinary abnormalities for this group. For age group 41 to 60 asymptomatic pyuria with other abnormalities constituted 46.66% & isolated pyuria constituted 33.33% of all

asymptomatic urinary abnormalities.For age groups 61 years and above asymptomatic pyuria with other abnormalities constituted 66.66% & isolated asymptomatic pyuria constituted 16.66% of all asymptomatic urinary abnormalities.As a whole asymptomatic pyuria constituted 26 cases out of 50 asymptomatic urinary abnormalities in this study constitute 52% of all asymptomatic urinary abnormalities and isolated asymptomatic pyuria constituted 17 cases of 50 asymptomatic urinary abnormalities constituted 34% of all asymptomatic urinary abnormalities.

## Causes of asymptomatic pyuria

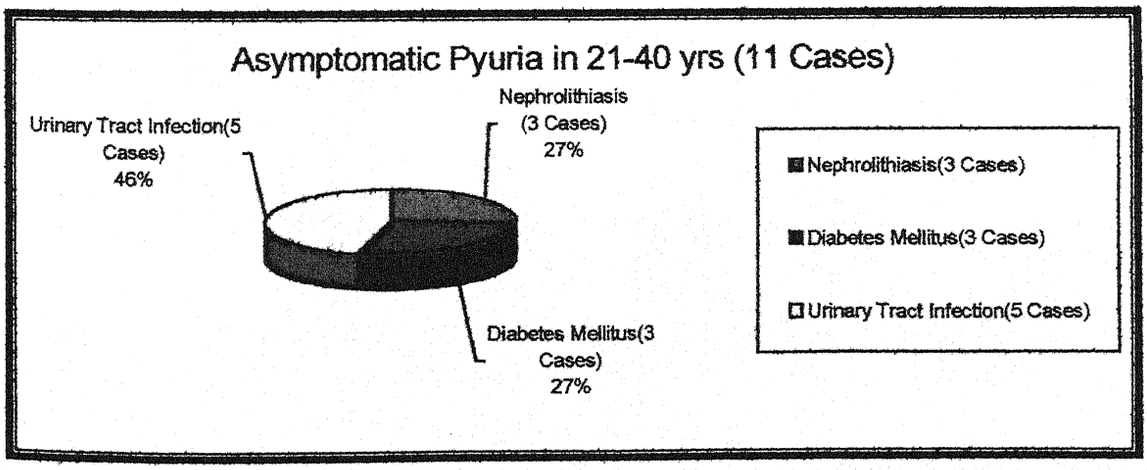
(a) Asymptomatic pyuria in 0-12 yrs (1 case)

In 0-12 yrs only one Male case of Asymptomatic Pyuria was detected and that was due to vesico ureteral reflux disease.

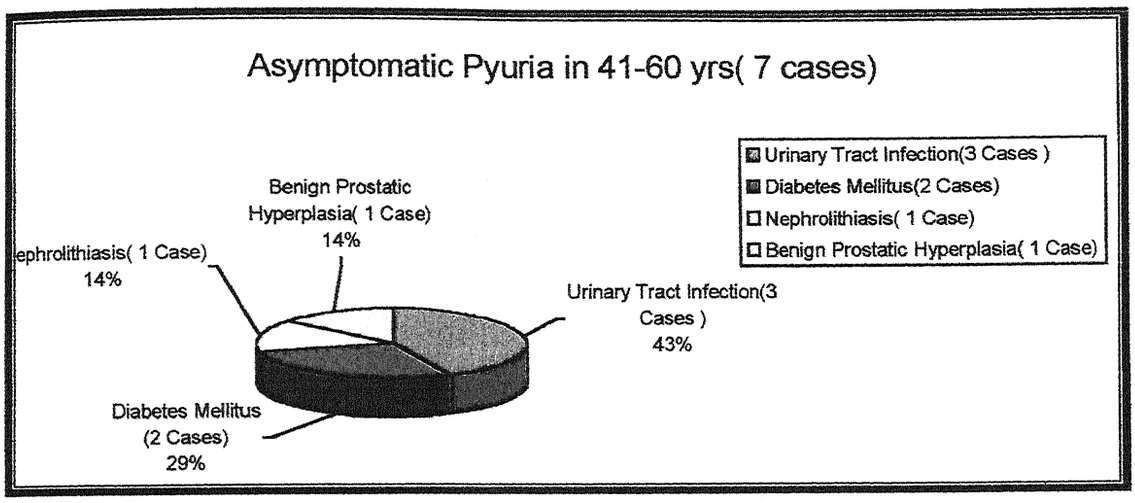
(b) Asymptomatic pyuria in 13-20 yrs (3 case)

There were 3 cases in this age group and all were due to urinary tract infection (1 was male, 2 were females)

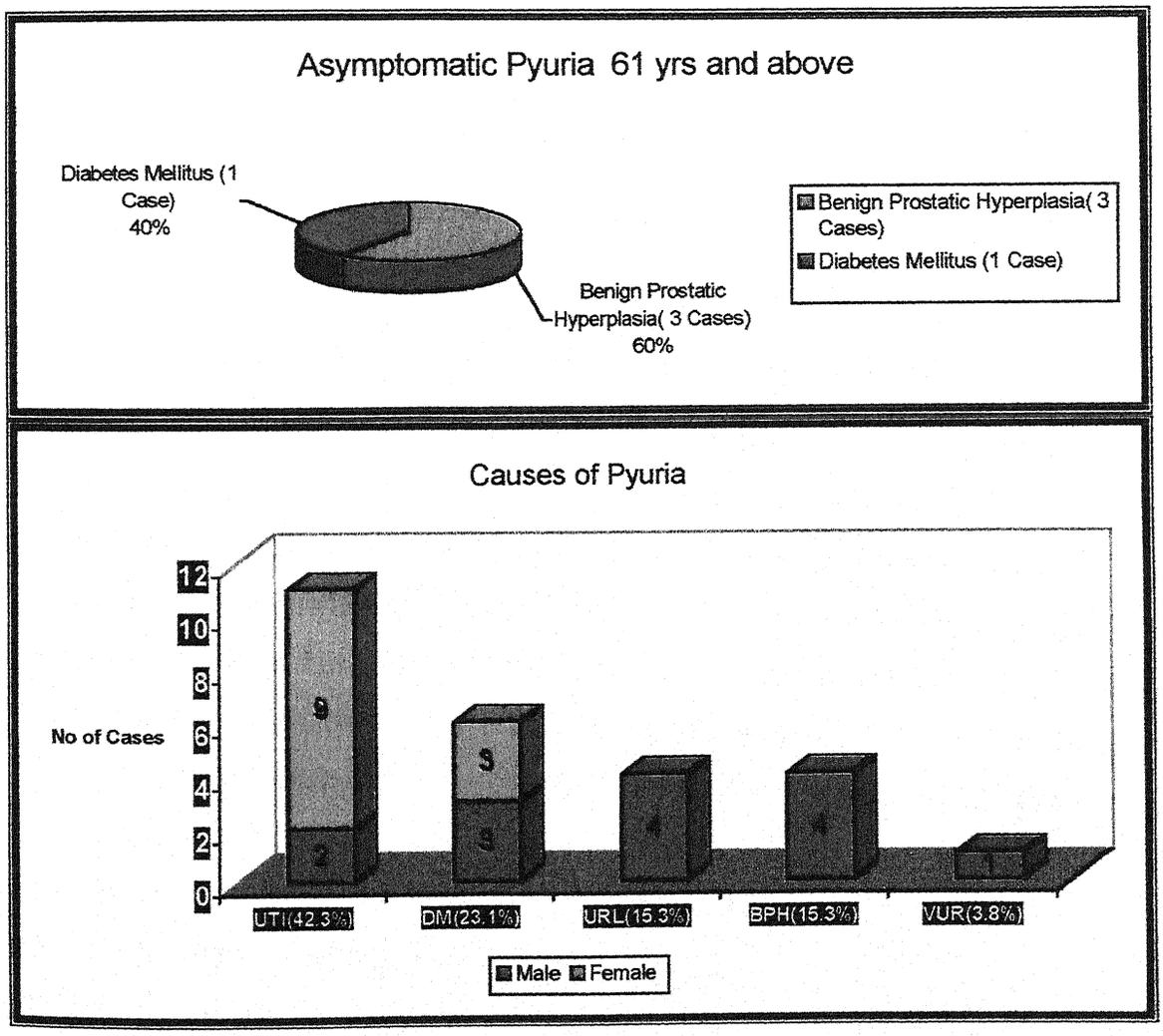
(c)



(d)


Asymptomatic Pyuria in 41-60 yrs( 7 cases)
Urinary Tract Infection(3 Cases )
Diabetes Mellitus(2 Cases)
Nephrolithiasis( 1 Case)
Benign Prostatic Hyperplasia( 1 Case)
Benign Prostatic Hyperplasia( 1 Case) 14%
ephrolithiasis( 1 Case) 14%
Urinary Tract Infection(3 Cases ) 43%
Diabetes Mellitus (2 Cases) 29%

(e)


Asymptomatic Pyuria 61 yrs and above
Diabetes Mellitus (1 Case) 40%
Benign Prostatic Hyperplasia( 3 Cases) 60%
Benign Prostatic Hyperplasia( 3 Cases)
Diabetes Mellitus (1 Case)
Causes of Pyuria
No of Cases
12
10
8
6
4
2
0
9
2
5
3
4
4
1
UTI(42.3%)
DM(23.1%)
URL(15.3%)
BPH(15.3%)
VUR(3.8%)
Male
Female

As shown in the above bar diagram , the causes of asymptomatic pyuria were urinary tract infection (42.3% , mostly females) , diabetes mellitus ( 23.1% with equal male female ratio) , urolithiasis ( 15.3% only in males) , benign prostatic hyperplasia (15.3% only in males) and vescicu ureteral reflux (3.8%only in males).

**( C) ASYMPTOMATIC GLYCOSURIA**

**Prevalence of Asymptomatic Glycosuria**

Table No. VI

PATIENTS DETECTED HAVING ASYMPTOMATIC GLYCOSURIA

| Age Group | Total Urinary Abnormalities | Glycosuria ( asymptomatic) | Isolated Glycosuria ( asymptomatic) |
|---|---|---|---|
| 0-12 | 04 | 01(25%) | 01(25%) |
| 13-20 | 03 | 00(0%) | 00(0%) |
| 21-40 | 22 | 03(13.6%) | 010(4.5%) |
| 41-60 | 15 | 02(13.33%) | 02(13.13%) |
| 61& above | 06 | 02(33.33%) | 00(0%) |
| **Total** | 50 | 08(16%) | 04(8%) |

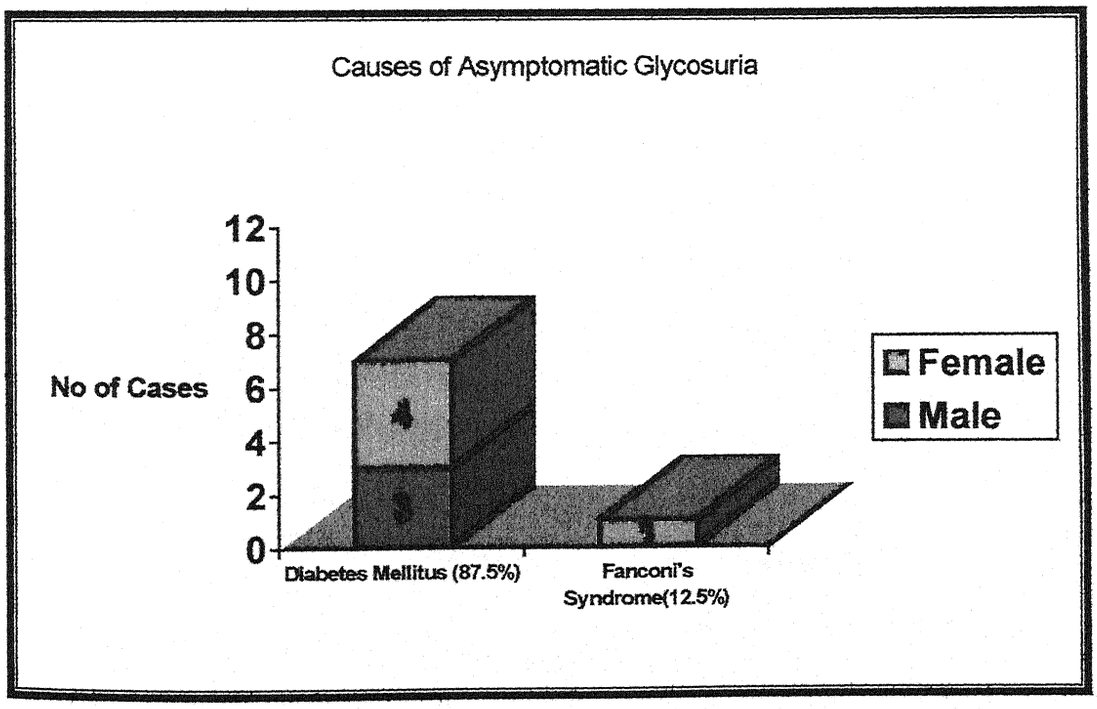
Asymptomatic Glycosuria occurred as isolated Glycosuria & as Glycosuria along with other urinary abnormalities, out of 50 cases of asymptomatic urinary abnormalities total case of asymptomatic Glycosuria were 8 making it 16% of all asymptomatic urinary abnormalities of which 4 were asymptomatic isolated glycosuria making it 5% of all asymptomatic urinary abnormalities.For different age group the prevalence of glycosuria with other urinary abnormalities and isolated glycosuria was as follows: For 0-12 Asymptomatic Glycosuria constituted 25% of all asymptomatic urinary abnormalities for that age group of which all occurred as isolated glycosuria. In age group 13-20 years no glycosuria was detected. For group 21-40 years Asymptomatic Glycosuria constituted 13.6% of all asymptomatic urinary abnormalities for that age group of which isolated glycosuria was 4.5 %. For group 41-60 years Glycosuria occurred only as

isolated glycosuria in this age group and prevalence was 13.13% of all of all asymptomatic urinary abnormalities for that age group. For above 61 - Glycosuria occurred only as glycosuria with other abnormalities & prevalence was 33.35%.

**Causes of Asymptomatic glycosuria**

| Age Group | Causes | Sex distribution |
|---|---|---|
| 0-12 | 1 fanconi's syndrome | Female |
| 13-20 | 0 | |
| 21-40 | 3DM | 2 Male, 1 Female |
| 41-60 | 2 DM | Both Females |
| 61& above | 2 DM | 1 Male , 1 Female |

All cases asymptomatic glycosuria / asymptomatic isolated glycosuria were attributed to Diabetic Mellitus except for 1 case which was due to fanconi's syndrome in the age group 0 – 12 years in this study the ratio of male: female was 3:4 respectively.




As shown in the above bar diagram , the major cause of asymptomatic glycosuria was diabetes mellitus ( 87.5% with slight female

predominance) with fanconi's syndrome making a small percentage ( 12.5% all females).

**(D) ASYMPTOMATIC CRYSTALLURIA**

**Prevalence of Asymptomatic Crystalluria**

Table No. VII

PATIENTS DETECTED HAVING ASYMPTOMATIC CRYSTALLURIA

| Age group | Total urinary abnormalities | Asymptomatic Crystalluria | Asymptomatic Isolated Crystalluria |
|---|---|---|---|
| 0-12 | 04 | 00(0%) | 00 (0%) |
| 13-20 | 03 | 00(0%) | 00(0%) |
| 21-40 | 22 | 05(22.72%) | 03(13.63%) |
| 41-60 | 15 | 01(6.66%) | 00(0%) |
| 61& above | 06 | 00(0%) | 00(0%) |
| **Total** | 50 | 06(12%) | 03(6%) |

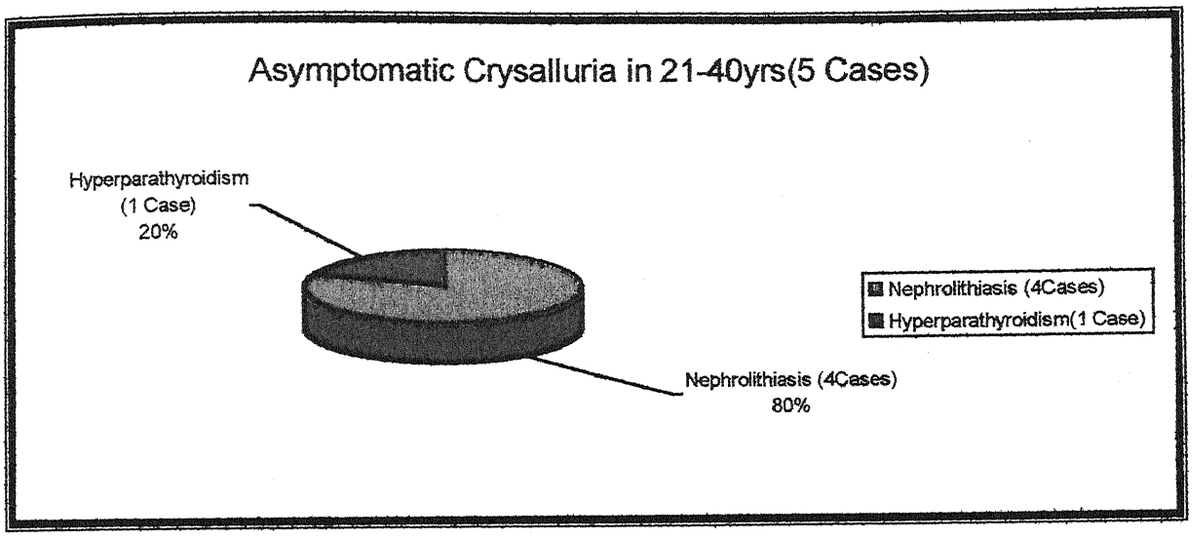
There were a total 6 cases of crystalluria and 3 cases of isolated crystalluria out of 50 asymptomatic urinary abnormalities , making them 12% & 6% respectively . No cases were seen in age group 0-12 & 13-20 year . Maximum 5 cases of asymptomatic crystalluria were seen in age group 21-40 years making it 22.72% of asymptomatic urinary abnormalities of that age group , out of which 13.63% occurred as isolated asymptomatic crystalluria . For age group 41-60 prevalence of asymptomatic crystalluria was 6.66% of all urinary abnormalities for that age group.

**Causes of Asymptomatic crystalluria**

(a) Asymptomatic Crystalluria 0-12yrs (0 cases)

(b) Asymptomatic Crystalluria 13-20 yrs (0 cases)

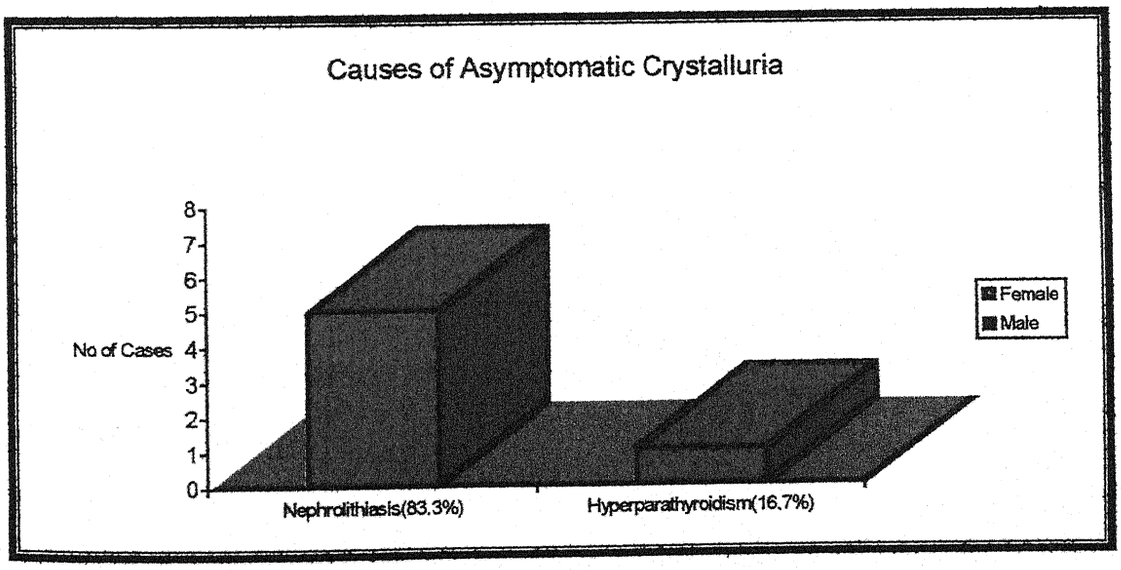
(c)




(d) Asymptomatic Crystalluria 41-60 yrs (1 cases)

One case of Asymptomatic Crystalluria (Male) was detected and this was caused by nephrolithiasis.

(e) Asymptomatic Crystalluria 61 yrs and above(0 cases)




As shown in the above bar diagram, the major cause of asymptomatic Crystalluria was Nephrolithiasis (83.3% all being males) while another cause hyperparathyroidism was also found (16.7% all cases being males).

## (E) HEMATURIA

### Prevalence of Asymptomatic hematuria

TABLE No. VIII

PATIENTS DETECTED HAVING ASYMPTOMATIC HEMATURIA

| Age group | Total urinary abnormalities | Hematuria | Isolated Hematuria |
|---|---|---|---|
| 0-12 | 04 | 01 (25%) | 00 (0%) |
| 13-20 | 03 | 00 (0%) | 00 (0%) |
| 21-40 | 22 | 02 (9.09%) | 01 (4.54%) |
| 41-60 | 15 | 01 (6.66%) | 00 (0%) |
| 61& above | 06 | 01 (16.66%) | 00 (0%) |
| **Total** | 50 | 05 (10%) | 01 (2%) |

### Causes of Asymptomatic hematuria

(a) Asymptomatic Hematuria in 0 – 12 years( 1 Case)

Only one case ( female ) was detected in this group and the cause was IgA Nephropathy.

(b) Asymptomatic Hematuria in 13 – 20 years( 0 Case)

(c) Asymptomatic Hematuria in 21 – 40 years( 2 Case)

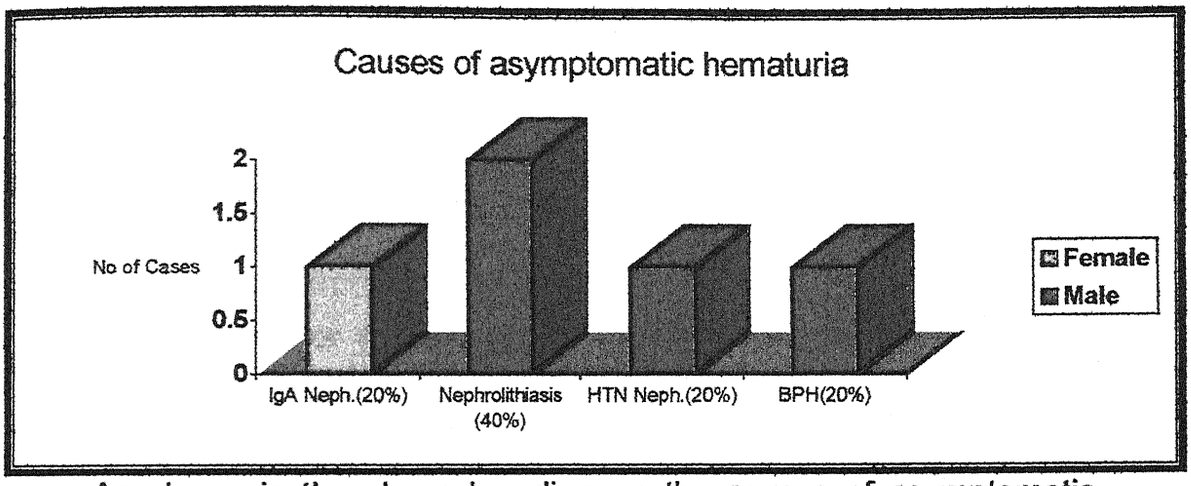
Both cases of asymptomatic hematuria in this group were males and both were caused by nephrolithiasis

( d) Asymptomatic Hematuria in 41 - 60years( 1 Case)

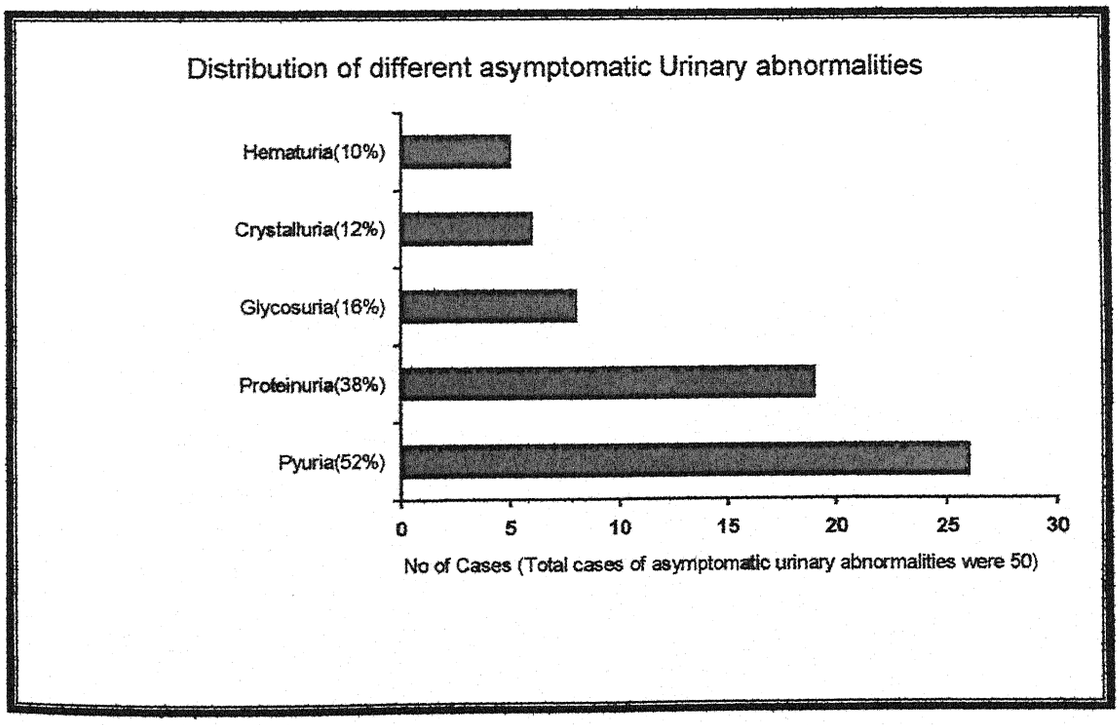
This male case of hematuria was due to Hypertensive Nephropathy.

(e) Asymptomatic Hematuria in 61 years and above( 1 Case)

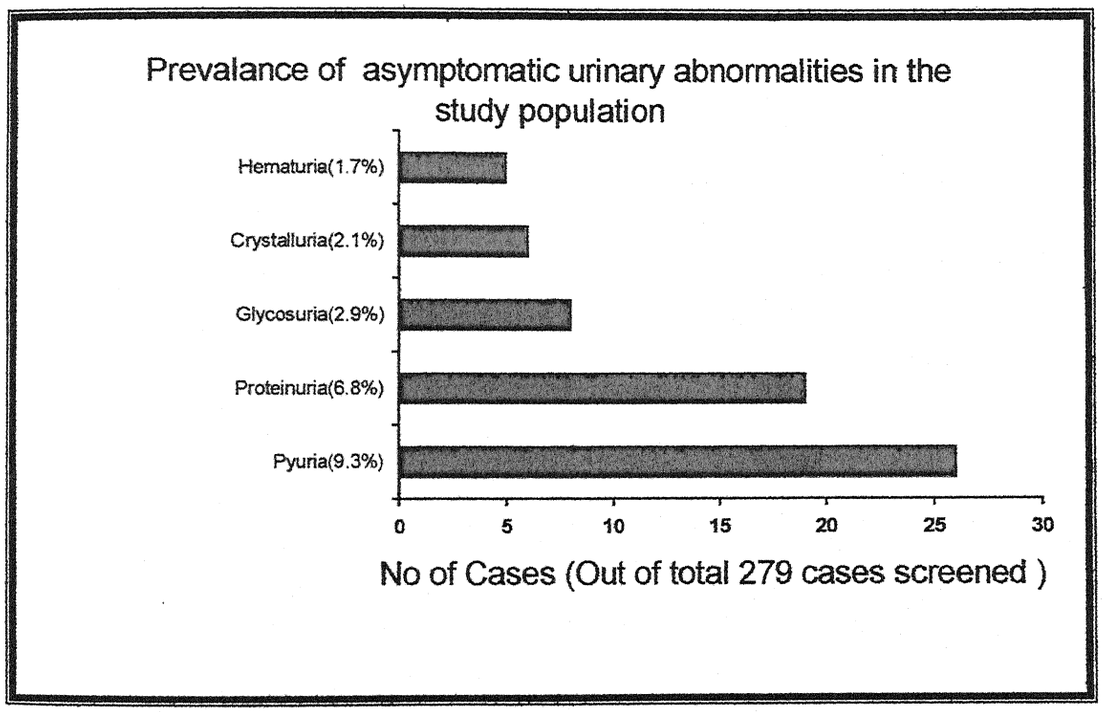
There was only one case of hematuria in this group and this was due to benign prostatic hypertrophy.




As shown in the above bar diagram the causes of asymptomatic hematuria were Nephrolithiasis (40%) , IgA Nephropathy(20%), hypertensive Nephropathy (20%) and benign prostatic hyperplasia ( 20 % ).



Amongst all different types of asymptomatic urinary abnormalities detected, asymptomatic Pyuria had the most common prevalence (52%), followed by asymptomatic Proteinuria(38%), Asymptomatic Glycosuria had prevalence of 16% , asymptomatic Crystalluria was 12% and asymptomatic Hematuria was 10%.




As shown in this chart above , out of 279 cases screened pyuria was seen in 9.3% cases, proteinuria was seen in 0.3%cases , glycosuria was seen 2.9%cases , crystalluria was seen in 2.1% cases and hematuria was seen in 1.7%cases .

# PREVALENCE OF ASYMPTOMATIC RENAL DISEASE

TABLE NO. I

AGE AND SEX DISTRIBUTION OF ASYMPTOMATIC DISEASES

| Age group | Asymptomatic diseases detected (No of Cases) | Sex distribution |
|---|---|---|
| 0-12 | 1 Fanconi's syndrome<br>1 Vesico ureteral reflux<br>1 FSGS<br>1 IgA Nephropathy | 1 female<br>1 male<br>1 female<br>1 female |
| 13-20 | 3 UTI | 2 Female , 1 male |
| 21-40 | 6 NPL (all males),<br>6 DM<br>5 UTI<br>3CGN | All males<br>3 Male, 3 Females<br>5 Females<br>3 Males |
| 41-60 | 1 NPL<br>5 DM<br>6 HTN Nephropathy<br>3 UTI<br>1 BPH<br>1 Renal Amyloidosis<br>1 Hyperparathyroidism | 1 Male<br>2 Males ,3 Females<br>4 Males, 2 Females<br>1 Male , 2 Female<br>1 Male<br>1 Male<br>1 Male |
| 61& above | 2 DM<br>3 BPH<br>1 HTN Nephropathy | 1 Male , 1 Female<br>3 Males<br>1 male |

As shown in table no. 1, there were various types of asymptomatic urinary abnormalities detected in different age groups, whose number and sex distribution varied with each age group . The above table shows age and sex distribution of all 50 cases of asymptomatic urinary abnormalities in this study.

## TABLE NO. II

## PREVALENCE OF ASYMPTOMATIC DISEASES IN THIS STUDY

| Disease | No. of asymptomatic cases detected | Ratio M:F | % of all asymptomatic Urinary abnormalities | % of total population screened |
|---|---|---|---|---|
| Diabetes Mellitus | 13 | 6:7 | 26% | 4.65% |
| Urinary tract infection | 11 | 2:9 | 22% | 3.94% |
| Nephrolithiasis | 07 | 7:0 | 14% | 2.51% |
| Hypertensive Nephropathy | 07 | 5:2 | 14% | 2.51% |
| Chronic Glomerulo Nephritis | 03 | 3:0 | 6% | 1.07% |
| Benign prostatic hyperplasia | 04 | 4:0 | 8% | 1.43% |
| Renal Amyloidosis | 01 | 1:0 | 2% | 0.35% |
| Hyperparathyroidism | 01 | 1:0 | 2% | 0.35% |
| IgA Nephropathy | 01 | 0:1 | 2% | 0.35% |
| Fanconi's syndrome | 01 | 0:1 | 2% | 0.35% |
| Focal segmental glomerulosclerosis | 01 | 0:1 | 2% | 0.35% |
| Vesico-ureteral reflux disease | 01 | 1:0 | 2% | 0.35% |

The above table no. II shows the presentation of various diseases that presented as asymptomatic urinary abnormalities in this study .The male to female ratio has been shown and the prevalence of each such disease has been shown in this table . The above table also shows the contribution which various diseases make to asymptomatic urinary

## PRESENTATION OF VARIOUS ASYMPTOMATIC DISEASES

### (A)Diabetes Mellitus

| Age group | No. of Cases | Presented as |
|---|---|---|
| 0-12 | 00 | None |
| 13-20 | 00 | None |
| 21-40 | 06 | 2 Gly.<br>2 Pr.<br>2 Gly.+ py. |
| 41-60 | 05 | 2 Gly.<br>2Py.<br>1 Pr. |
| 61& above | 02 | 1 Gly.+py.<br>1 Gly.+pr. |

Out of Total 13 cases = 4 presented as Glycosuria( 30.76%) ; 3 presented as Proteinuria( 23.07%) ; 2 presented as Pyuria( 15.38%) ; 3 presented asGlycosuria +Pyuria(23.07%) ; 1 Pesented as Glycosuria +Proteinuria(7.7%). Most common presentation was as glycosuria(30.76%) and least common was as glycosuria + proteinuria(7.7%).

### (B) Urinary tract infection

| Age group | No of case | Presented as |
|---|---|---|
| 0-12 | 00 | None |
| 13-20 | 03 | All Py. |
| 21-40 | 05 | All Py. |
| 41-60 | 03 | All Py. |
| 61& above | 00 | None |

Total 11 cases of asymptomatic Urinary tract infection of were detected in this study and all ( 100% ) of them presented as pyuria .

## (C) NEPHROLITHIASIS

| Age group | No of case | Presented as |
|---|---|---|
| 0-12 | 00 | None |
| 13-20 | 00 | None |
| 21-40 | 06 | 2 ox.<br>1 ox. +py.<br>1 ox.+py.+He.<br>1 He.<br>1 Py. |
| 41-60 | 01 | Py.+ox.+ph. |
| 61& above | 00 | None |
| Total | 07 | 2 Ox. ;<br>1 Ox.+ Py.<br>1 Ox+Py.+He<br>1 Ox.+Py.+Ph.<br>1 He. |

Total 7 cases detected = of which 2 presented as calcium oxalate crystalluria(28.57%) ; 1 as oxalate + pyuria(14.28%) ; 1 as oxalate + pyuria + hematuria(14.28%) ; 1 as oxalate + hematuria + phosphate crystals(14.27%) ; and 1 as isolated hematuria(14.27%).Most common presentation was as asymptomatic calcium oxalate crystals Other presentations were calcium oxalate crystals with other abnormalities like hematuria , pyuria and phosphate crystals .

## (D) HTN Nephropathy

| Age Group | No of Case | Presented as |
|---|---|---|
| 0-12 | None | - |
| 13-20 | None | - |
| 21-40 | None | - |
| 41-60 | 06 | 5 as pr.<br>1 as Pr.+ He. |
| 61& above | 01 | 1 as Pr. |
| Total | 07 | 6 as pr.<br>1 as Pr. + He. |

Total 7 cases detected= 6 presented as proteinuria(85.77%) and 1 presented as proteinuria with hematuria(14.23%). Most common presentation was as proteinuria .

**( E) Chronic glomerulo nephritis**

| Age Group | No of Case | Presented as |
|---|---|---|
| 21-40 | 03 | All as pr. |

Total 3 cases detected and all (100%) presented as proteinuria .

**(F) Benign prostatic hypertrophy**

| Age Group | No of Cases | Presented as |
|---|---|---|
| 41-60 | 01 | 1 as Py. |
| 61 & above | 03 | 1 Py.<br>1 Py. + Pr.<br>1Py. + He. |
| Total | 04 | 2 Py.<br>1 Py. +pr.<br>1 Py. + He. |

Total 4 cases detected = 2 presented as pyuria ( 50 %) ; 1 as pyuria + proteinuria ( 25%) ; and 1 as proteinuria + hematuria ( 25 % ). Most common presentation was as pyuria.

**(G) Renal Amyloidosis**

| Age Group | No of cases | Presented as |
|---|---|---|
| 41-60 | 01 | Pr. |

Only one case seen in this study that presented as proteinuria(100%) .

**(H) Hyperparathyroidism**

| Age Group | No of cases | Presented as |
|---|---|---|
| 21-40 | 01 | Ox. |

Only 1 case of asymptomatic hyperparathyroidism detected that presented as calcium oxalate crysytalluria( 100 %)

**(I) IgA Nephopathy**

| Age Group | No of cases | Presented as |
|---|---|---|
| 0-12 | 01 | Pr.+He. |

Only 1 case seen that presented as proteinuria with hematuria (100%)

**( J) Fanconi's syndrome**

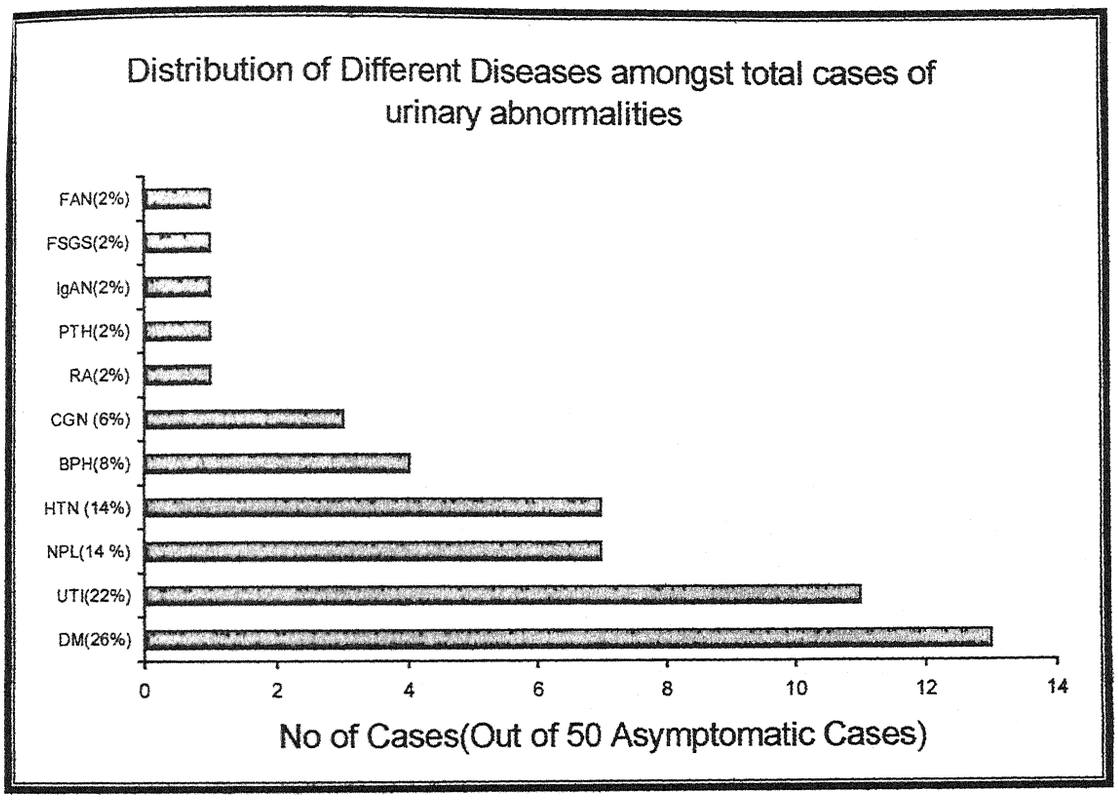
| Age Group | No of cases | Presented as |
|---|---|---|
| 0-12 | 01 | Gly. |

Only 1 case seen that presented as Glycosuria (100%).

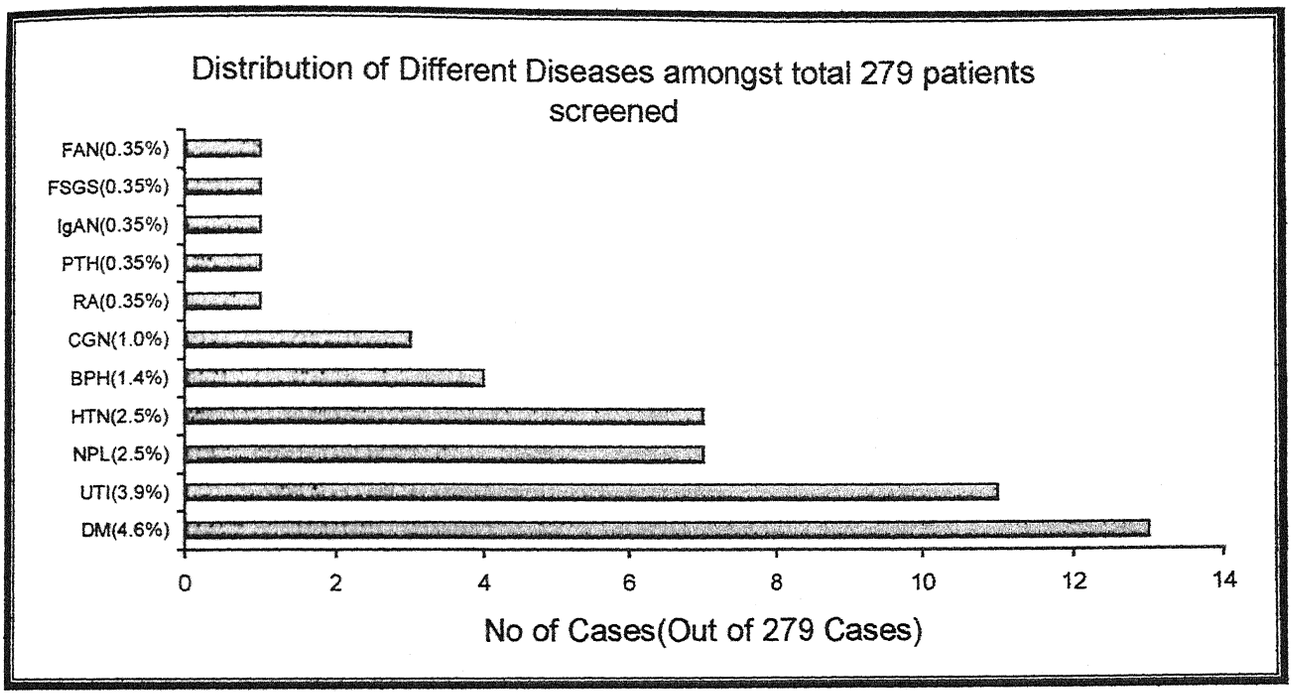
**(K) Focal segmental glomerulosclerosis**

| Age Group | No of cases | Presented as |
|---|---|---|
| 0-12 | 01 | Pr. |

Only 1 case seen that presented as proteinuria (100%) .




As shown in the above table , Diabetes mellitus ( DM) was seen as 26% of all asymptomatic urinary abnormalities , Urinary tract infection ( UTI) was seen as 22% of all asymptomatic urinary abnormalities, Nephrolithiasis(NPL) was seen as 14% of all asymptomatic urinary abnormalities, Hypertension (HTN) was seen as 14% of all asymptomatic urinary abnormalities, Benign prostatic hyperplasia(BPH) was seen as 8% of all asymptomatic urinary abnormalities , Chronic glomerulonephritis ( CGN) was seen as 6% of all asymptomatic urinary abnormalities , Renal amyliodosis (RA) as 2% , Hyperparathyroidism (PTH) as 2% , IgA Nephropathy( IgA N) as 2% , Focal segmental glomerulosclerosis (FSGS) as 2% and Fanconi's syndrome ( FAN) as 2% of all asymptomatic urinary abnormalities.




As shown in the above table , Diabetes mellitus ( DM) was seen in 4.6% all people screened , Urinary tract infection ( UTI) was seen in 3.9% of all people screened, Nephrolithiasis(NPL) was seen in 2.5% of all people screened, Hypertension (HTN) was seen in 2.5% of all people screened, Benign prostatic hyperplasia(BPH) was seen in 1.4% of all people screened , Chronic glomerulonephritis( CGN) was seen in 1% of all people screened, Renal amyliodosis(RA) in 0.35% , Hyperparathyroidism (PTH) in 0.35% , IgA Nephropathy( IgA N) in 0.35% , Focal segmental glomerulosclerosis (FSGS) in 0.35% and Fanconi's syndrome ( FAN) in 0.35% of all people screened.

# DISCUSSION

# DISCUSSION

This study was conducted in Bundelkhand region with the help of renal disease detection camps. In these camps, all age and sex groups patients attending the renal camps were screened by urine routine and microscopic examination.

A total of 279 patients of various age and sex groups were screened by urine examination .Of these 279 cases, 172 were males and 107 were females making a male to female ratio of 1.6:1. For convenience of screening, the study population was divided into discrete study groups according to age. The maximum number of patients who attended the renal camps belonged to the age group 21-40 years and minimum to 61and above years group.

A very similar study has been done by N. Vidya Acharya et al. This study conducted in the city of Bombay involved the study of 430 subjects. A similar division according to age was also done in this study. In addition in this study done by N. Vidya acharya et al, the subjects attending the renal camps were divided further into low socio economic group and middle socio economic group. In the present study in Bundelkhand region however, no such division could be been done, because almost all patients who attended our renal camps belonged to the low economic group thus the scope for comparison was minimal.

In the study asymptomatic urinary abnormalities in Bundelkhand region, out of 279 patients screened by urine routine microscopy examination 68 patients turned out to have detectable urinary abnormality

in their urine. These included symptomatic cases, follow up cases and fresh asymptomatic cases. These 68 cases out of 279 made up 24.37%. In the study of N. Vidya acharya et al , out of 430 patients screened , 151 had detectable urinary abnormalities , this made up 45.5% of the study population . Thus as compared to this 45% of detectable urinary abnormality in the Bombay study, the present study here in Bundelkhand region had 24.37% of detectable urinary abnormality. These 24.37% cases of urinary abnormality included proteinuria, hematuria, Pyuria, crystalluria and Glycosuria. In the study conducted in Bombay however, the only urinary abnormalities stressed on were hematuria, proteinuria and ketonuria, while pyuria and crystalluria were not screened for. In the study done by N.Vidya Acharya et al, the method used to detect asymptomatic urinary abnormalities was dipsticks examination of urine, while method used the present study here was routine and microscopic examination of urine.

Amongst the 68 patients out of 279 detected to have urinary abnormalities, 50 (17.9%) cases were asymptomatic .The maximum prevalence of patients with asymptomatic urinary abnormalities was seen in the age group 61 and above (30%) and the minimum prevalence in 13-20 years age group (11.11%). In this study of 279 people 143 were males and out of these 29 had asymptomatic urinary abnormalities (20.27%), while 86 were females out of which 21 were having asymptomatic urinary abnormalities (24.11%). Thus females were found to have asymptomatic urinary abnormalities a little more frequently as compared to males. In males asymptomatic urinary abnormalities were most prevalent in the age group 61 and above but maximum cases were seen in 21-40 years age group, while in females it was most prevalent in the group 41-60 years with maximum number of cases in 21-40 years

group . Minimum prevalence was seen in 13-20 years age group in both sexes.

Amongst various asymptomatic urinary abnormalities , asymptomatic proteinuria was found in 19 cases out of 50 cases of asymptomatic urinary abnormalities thus making 38 % of this group (19 out of 50). Out of these 19 patients, 14 had isolated proteinuria making 28% asymptomatic urinary abnormalities (14 out of 50). Considering these 19 cases of proteinuria and 14 cases of isolated proteinuria as a percentage of the whole population screened (279 people) , proteinuria was seen in 6.9% (19 out of 279) and isolated proteinuria in 5% ( 14 out of 279) . In the pediatric age group 0-12 years number of cases of proteinuria were 2 and number of total people screened in this age group were 25 ,thus the prevalence of proteinuria was 8% (2 out of 25) in this study as compared to 4% in the study done by Pygia M.J. , Lott JA et al who studied 6197 school children in 1974 in Japan .In the study done by N. Vidya acharya et al , the prevalence of proteinuria was 23.2% in the whole study group and of isolated proteinuria was 7.2% . Amongst the causes of asymptomatic proteinuria, Hypertension was seen in 42.1% , Diabetes material in 21.2% ,Chronic glomerulo nephritis in 15.8% , Renal Amyloidosis in 5.2% , Benign prostatic hyperplasia in 5.2% , Focal segmental glomerulosclerosis in 5.2% and IgA Nephropathy in 5.2% cases of Asymptomatic proteinuria.

Asymptomatic Pyuria was detected in 26 patients out of 50 patients with asymptomatic abnormality making 52% (26 out of 50). Out of there 26 patients, 17 had isolated pyuria making 34% (14 out of 50). Maximum number of Pyuria in both sexes was seen in the age group 21-40 years (sexually active group). Amongst all the causes of Asymptomatic pyuria, urinary tract infection was seen in 42.3% (11 out of 26) , Diabetes mellitus

in 23.1% (6 out of 26) , Nephrolithiasis in 15.3% ( 4 out of 26) , Benign prostatic hyperplasia in 15.3% (4 out of 20) and Vesico ureteral reflux in 3.8% ( 1 out of 26) . In the study of N.V. Acharya et al, pyuria was not screened for and nor has it been screened in other studies, so a comparison could not be made as to what prevalence of asymptomatic pyuria is there in other parts of the country or world.

Asymptomatic Glycosuria was detected in 8 cases out of all patients (50) of asymptomatic urinary abnormabilities , thus it was 16% (5 out of 50) while isolated glycosuria made up 8%( 4 out of 50 )of all asymptomatic urinary abnormalities . Majority of cases detected were in age group 21-40 and 41-60 years but maximum prevalence was for the age group 61 and above. Only 1 case was seen in 0-12 years (pediatric) age, and the cause was Fanconi's syndrome. In all other cases, the cause of Glycosuria turned out to be Diabetes mellitus. Therefore causes of glycosuria were, Diabetes mellitus 87.5% (7 out of 8) and Fanconi 's syndrome 12.5% ( 1 out of 8) .Glycosuria when considered as a fraction of the whole population screened , was found to be 2.86% ( 8 out of 279) . The study of V.N. Acharya et al showed a prevalence of glycosuria in 4.4%.

Asymptomatic Crystalluria was detected in 6 cases out of 50 cases, thus prevalence was 12% (6 out of 50) .Maximum number of cases were detected in the age group 21-40 years and was exclusively detected in male population in this study. Out of all cases asymptomatic crytalluria detected (6) majority were due to Nephrolithiasis 83.3% ( 5 out of 6) and another less frequent cause was Hyperparathyroidism 16.7%( 1 out of 6).

Amongst all cases of Asymptomatic urinary abnormalities (50), hematuria was seen in 5 cases thus making 10% (5 out of 50) and isolated

hematuria was seen in 1 case 2% ( 1 out of 50). Maximum number of cases of hematuria were seen in the 21-40 years age group (3 cases), but the prevalence was most in pediatric age group (25% of all asymptomatic urinary abnormalities. Considering it as a percentage of total population screened the prevalence was 1.79 %( 5 out of 279). In the pediatric age group the prevalence was 8% (1 out of 25 cases). In the study by Vidya N Acharya et al, the prevalence of isolated proteinuria was found out to be 3.9% and that of asymptomatic hematuria with proteinuria was found out to be 16.6% . Amongst the causes of Asymptomatic hematuria , Nephrolithiasis was seen in 40 % ( 2 out of 5 ) , while IgA Nephropathy , Hypertensive nephropathy , and Benign prostatic hyperplasia made 20% each ( 1 out 5 cases each ) .

Discussing all Asymptomatic urinary abnormalities together, it was seen that , out of 50 cases of Asymptomatic urinary abnormalities 26 had Asymptomatic pyuria thus making 52% of all Asymptomatic urinary abnormalities , 19 had asymptomatic proteinuria making 38% of all Asymptomatic urinary abnormalities , 8 had asymptomatic glycosuria thus making 16% of all Asymptomatic urinary abnormalities , 6 had asymptomatic crystalluria thus making 12% of all Asymptomatic urinary abnormalities , 5 had asymptomatic hematuria making 10 % of all Asymptomatic urinary abnormalities.

Considering each type of Asymptomatic urinary abnormalities as a percentage of the whole population screened , pyuria was found in 9.3% of all cases ( 26 out of 279) , proteinuria in 6.8% ( 19 out of 279) , glycosuria in 2.8% ( 8 out of 279) , crystalluria in 2.15% ( 6 out of 279) and hematuria in 1.79% ( 5 out of 279). A variety of diseases were detected as causes of

asymptomatic urinary abnormalities in this study the details of which has been discussed as below.

Of all diseases, there were 13 cases of Diabetes mellitus of which 6 were males and 7 were females. Thus out of 50 cases of asymptomatic cases detected 13 were due to diabetes making 26 %( 13 out of 50). Considering Diabetes in context to the whole study group, it made up 4.65 %( 13 out of 279). Out of Total 13 cases 4 presented as Glycosuria( 30.76%) ; 3 presented as Proteinuria( 23.07%) ; 2 presented as Pyuria( 15.38%) ; 3 presented as Glycosuria +Pyuria(23.07%) ; 1 Presented as Glycosuria +Proteinuria(7.7%). Most common presentation was as glycosuria(30.76%) and least common was as glycosuria + proteinuria(7.7%).

Another commonly detected disease as cause of asymptomatic urinary abnormalities was asymptomatic urinary tract infection .There were total 11 cases of asymptomatic Urinary tract infection detected. Out of these 2 were males and 9 were females. Thus Urinary tract infection was 22% of all asymptomatic urinary abnormalities (11 out of 50 cases). Considering asymptomatic Urinary tract infection in the whole study population, it made up 3.94 %( 22 out of 279 cases). Total 11 cases of asymptomatic Urinary tract infection of were detected in this study and all (100%) of them presented as pyuria.

There were 7 cases of asymptomatic Nephrolithiasis detected in this study. Out of these all of them were males. Thus asymptomatic Nephrolithiasis made up 14% (7 out of 50 cases) of asymptomatic urinary abnormalities. Nephrolithiasis was seen in 2.51% of total people screened (7 out of 279) .Total 7 cases of Nephrolithiasis were detected , of which 2

presented as calcium oxalate crystalluria(28.57%) ; 1 as oxalate crystals + pyuria (14.28%) ; 1 as oxalate crystals + pyuria + hematuria(14.28%) ; 1 as oxalate crystals + hematuria + phosphate crystals(14.27%) ; and 1 as isolated hematuria (14.27%) .Most common presentation was as asymptomatic calcium oxalate crystals. Other presentations were calcium oxalate crystals with other abnormalities like hematuria, pyuria and phosphate crystals.

Out of 50 cases of asymptomatic abnormalities, Hypertensive nephropathy was detected in 7 cases. Out of these 5 were males and 2 were females. Thus Hypertensive nephropathy made up 14% (7 out of 50) of all asymptomatic urinary abnormalities. Hypertensive nephropathy was seen in 2.51% (7 out of 279) of total patients screened. Of total 7 cases detected, 6 presented as proteinuria (85.77%) and 1 presented as proteinuria with hematuria (14.23%). Most common presentation was as proteinuria.

There were 3 cases of chronic glomerulonephritis seen out of 50 cases thus making 6% (3 of 50) of all asymptomatic urinary abnormalities. All cases were males. It was seen in 1.07% (3 out of 279 cases) of the study population. Total 3 cases detected and all (100%) presented as proteinuria.

There were 4 cases of asymptomatic Benign prostatic hyperplasia making 8% (4 out of 50) of all asymptomatic urinary abnormalities. All these cases were males. Considering it in the total population screened, it was seen in 1.43% of the total study population (4 out of 279). Total 4 cases detected 2 presented as pyuria ( 50 %) ; 1 as pyuria + proteinuria (

25%) ; and 1 as proteinuria + hematuria ( 25 % ). Most common presentation was as pyuria.

Only 1 case of Renal Amyloidosis was seen, thus making 2% of all asymptomatic urinary abnormalities (1 out of 50). This case was male. Considering the total study population, Renal Amyloidosis was seen in 0.35%( 1 out of 279) . This case presented as proteinuria.

Only 1 case of Hyperparathyroidism was seen, thus making 2% of all asymptomatic urinary abnormalities (1 out of 50). This case was male. Considering the total study population, Hyperparathyroidism was seen in 0.35 %( 1 out of 279). This case presented as oxalate crysatalluria.
Only 1 case of IgA Nephropathy was seen, thus making 2% of all asymptomatic urinary abnormalities (1 out of 50) .This case was female. Considering the total study population, IgA Nephropathy was seen in 0.35 %( 1 out of 279). This case presented as hematuria and proteinuria.

Only 1 case of Fanconi's syndrome was seen, thus making 2% of all asymptomatic urinary abnormalities (1 out of 50). This case was female. Considering the total study population, Fanconi's syndrome was seen in 0.35 %( 1 out of 279) . This case presented as glycosuria.

Only 1 case of Focal segmental glomerulosclerosis was seen, thus making 2% of all asymptomatic urinary abnormalities (1 out of 50). This case was female. Considering the total study population, Focal segmental glomerulosclerosis was seen in 0.35 %( 1 out of 279). This case presented as proteinuria.

Only 1 case of Vesicuo ureteral reflux disease was seen, thus making 2% of all asymptomatic urinary abnormalities (1 out of 50). This case was female. Considering the total study population Vesicuo ureteral reflux disease was seen in 0.35 %( 1 out of 279). This case presented as pyuria.

# SUMMERY & CONCLUSION

# SUMMARY & CONCLUSION

To summarize, this study which was conducted in Maharani Laxmi Bai medical college, Jhansi with the help of frequently organized organized Renal diseases detection camps in various regions of Bundelkhand region, found out that :

1. Asymptomatic urinary abnormalities were not as infrequent as is generally thought.

2. Of the total 279 patients screened by urine routine microscopy in renal camps, Asymptomatic urinary abnormalities were detected in 17.9% people, who never knew or had any symptoms of renal disease .

3. Various Asymptomatic urinary abnormalities detected in this study were Proteinuria, Pyuria , Hematuria , Crystalluria and Glycosuria .

4. Most commonly prevalent Asymptomatic urinary abnormality in this study was Asymptomatic Pyuria .It was found in 9.3% cases screened. Most common detected cause of asymptomatic pyuria was urinary tract infection. Others causes were Diabetes Mellitus, Nephrolithiasis , BPH and Vesico ureteral reflux disease( uncommon).

5. Asymptomatic Proteinuria was found in 6.8% people screened. Isolated proteinuria was found in 5% of population. Most common cause of proteinuria was Hypertensive nephropathy. Other common causes of

Asymptomatic proteinuria were Diabetes Mellitus and Chronic glomerulo nephritis.

6. Asymptomatic Glycosuria was detected in 2.9% of the population screened . Most common cause of asymptomatic Glycosuria was Diabetes Mellitus with an almost equal distribution in both sexes .

7. Asymptomatic Crystalluria was detected in 2.1% of population screened. It was found exclusively in male population and was most commonly associated with Nephrolithiasis.

8. Asymptomatic Hematuria was detected in 1.7% of the total patients screened while isolated Hematuria was detected in 0.35%. Most common cause of Asymptomatic Hematuria in this study was Nephrolithiasis (found as cause in 40% cases of asymptomatic hematuria detected). While IgA Nephropathy Hypertensive nephropathy and Benign prostatic hyperplasia were other causes.

9. During follow up study of cases detected to have asymptomatic urinary abnormalities, various modalities of investigations were used to reach a conclusive diagnosis for each case . A variety of diseases were detected as cause of these asymptomatic urinary abnormalities , which included Diabets Mellitus, Hypertension , Urinary tract infection , Nephrolithiasis, Benign prostatic hyperplasia , Chronic glomerulonephritis, Renal Amyloidosis , Hyperparathyroidism , IgA Nephropathy , Focal segmental glomerulosclerosis and Fanconi's syndrome.

10. Asymptomatic Diabetes Mellitus was most commonly detected cause of Asymptomatic urinary abnormalities in this study contributing 26% of all Asymptomatic urinary abnormalities detected. Asymptomatic Diabetes mellitus was detected in 4.6% of the population screened.

11. Asymptomatic urinary tract infection was not uncommon and was detected in 3.94% of all cases screened with a male: female ratio of 2:9.

12. Nephrolithiasis and Hypertensive Nephropathy were each detected in 2.5% of total population screened with a male predominance in both diseases.

13. Chronic Glomerulonephritis and Benign Prostatic hyperplasia were detected in 1.07% and 1.43% of the population screened respectively. Both of there diseases showed male predominance .

14. Renal Amyloidosis, Hyperparathyroidism, IgA Nephropathy, Fanconi's Syndrome , Focal segmental Glomerulosclerosis and vesciculo ureteral reflux were other diseases detected in this study as causes of Asymptomatic urinary abnormalities each detected in 0.35% of the total population screened.

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# MASTER CHART

# MASTER CHART

## PART I -RESULTS OF RENAL DISEASE DETECTION CAMPS

| Serial No. | Name | Age/Sex | Blood Pressure in mmHg | Clinical features | Urine Routine/microscopy | Blood Sugar | Fundus |
|---|---|---|---|---|---|---|---|
| 1 | Smt. Neelima.Shukla | 55/F | 140/100 | Hypothyroidism. | NAD | -95mg%(R) | Not done |
| 2. | Shanti Bai | 60/F | . 100/50 | FUC/CRF/HTN/ RA 40 Swelling Abdominal pain | Protein ++ | 72%mg(4/npp) | B/L Cataract |
| 3. | Mehmooda | 32/F | 110/ 70 | FUC. Renal Stone. | NAD | not done | not done |
| 4. | Smt.Sushama Shivhara | 32/F | 108/76 | Burning micturition | Pus cells occasional Rest NAD. | Not done | Not done |
| 5. | Hamida | 48/F | 100/74 | none | NAD | Not done | Not done |
| 6. | Moh Hanif farook | 50/M | 130/84 | none | NAD | Not done | Not done |
| 7. | Km. Aasha | 18/F | 120/74 | Pain in Rt. renal angle 3 days | NAD | Not done | Not done |
| 8. | Ram deen | 60/M | 144/76 | Burning micturition ↑ frequency | NAD | Not done | Not done |
| 9. | Panku | 55/M | 134/84 | Lump in left i.f. | NAD | Not done | Not done |
| 10. | Ramdol | 64/M | 156/98 | none | Sugar++++ Pus cells occasional. | Not done | Not done |
| 11. | Rama malvaya | 38/F | 126/84 | constipation | NAD | Not done | Not done |
| 12. | Vani | 50/F | 110/70 | | Pus Cells 3-4/hpf Epith occ. | Not done | Not done |
| 13. | Ghanghey | 37/M | 120/80 | Epigastric pain | Pus cell 1-2 | Not done | Not done |
| 14. | Anjana | 36/F | 110/74 | None | Sugar+++ pus cells 12 – 14 /hpf | 296mg% 2h pp | Not done |
| 15. | Panveer | 55/F | 120/70 | None | NAD | Not done | Not done |
| 16. | Raj Kumari | 38/F | 124/80 | None | NAD | Not done | Not done |
| 17. | Pram Giri | 60/M | 110/78 | None | NAD | Not done | Not done |
| 18. | Davandra | 36/M | 108/66 | None | NAD | Not done | Not done |
| 19. | Dawarika | 49/M | 140/80 | Left lain pain | NAD | Not done | Not done |
| 20. | Mahd Muskiq | 51/M | 156/86 | Polydypsia polyurea hesitancy | Sugar ++++ Pus cells 6-8 / hpf | Not done | Not done |
| 21. | Smt. Pankuwar | 44/F | 120/78 | none | NAD | Not done | Not done |
| 22. | Ajay Srivastava | 40/M | 136/90 | Known DM | NAD | 83mg% 2hrpp | |
| 23. | Maya Devi | 22/F | 146/90 | Back ache | NAD | Not done | Not done |
| 24. | Radharaman. | 45/M | 110/68 | loin pain | Cast hyaline. Rest NAD | Not done | Not done |
| 25. | Shyam Kishor | 46/M | 124/86 | Difficulty micturition | NAD | Not done | Not done |
| 26. | Suresh Chandra | 44/M | 142/90 | ↑ frequency Burning mic. | NAD | Not done | Not done |
| 27. | Nagma | 31/F | 122/70 | None | NAD | Not done | Not done |

| | | | | | | | |
|---|---|---|---|---|---|---|---|
| 28. | Khilan | 30/M | 100/60 | None | NAD | Not done | Not done |
| 29. | Kusum | 35/F | 106/80 | None | NAD | Not done | Not done |
| 30. | Anjana | 36/F | 114/68 | None | NAD | Not done | Not done |
| 31. | Kirti | 10/F | 106/60 | None | Urine protein ++ | Bl. Sugar 94mg%(R) | Not done |
| 32. | L.R. Verma | 46/M | 102/70 | None | Pus cell occasional. Phosphate crystals | Not done | Not done |
| 33. | RamKale | 60/M | 100/70 | Pain in Knee j Ghabrahat | NAD | Not done | Not done |
| 34. | Bhagowerti | 40/F | 130/80 | Arthralgia insomnia Anorexia | Pus cell present | Bl. Sugar 341 mg% (R) | Fundus DR + |
| 35. | Hadid Khan | 65/M | 160/90 | None | Protein++ pus cells 6-8 /hpf | 77mg% | Not done |
| 36. | Rana Kaheed | 40/F | 140/80 | None | NAD | Not done | Not done |
| 37. | Mohan | 40/M | 120/86 | None | NAD | Not done | Not done |
| 38. | Nasreem | 33/F | 126/76 | Recurrent UTI | NAD | Not done | Not done |
| 39. | Smt. BD. Singh | 53/F | 100/70 | None | NAD | Not done | Not done |
| 40. | K.P. Singh | 62/M | 154/90 | None | NAD | Not done | Not done |
| 41. | Smt. Kapoori | 42/F | 180/80 | None | NAD | Not done | Not done |
| 42. | Mohd. mushtaque | 51/M | 140/80 | Pain during micturition | Pus cells +++ (Field full) | Bl. Sugar 94mg% | Not done |
| 43. | Lakshmi | 42/F | 120/76 | None | NAD | Not done | Not done |
| 44. | Smt. Sajeeda | 45/F | 144/86 | None | NAD | Not done | Not done |
| 45. | Raj Kumari | 35/F | 128/80 | Known pt. of DM | Protein ++ Sugar Nil | Sugar 154mg% 4hrpp | Not done |
| 46. | Nolha Devi | 50/F | 110/70 | None | NAD | Not done | Not done |
| 47. | Mohan | 40/M | 100/80 | Gen. swelling 5 yr. | NAD | Not done | Not done |
| 48. | Rajan sharma | 40/M | 146/80 | None | NAD | Not done | Not done |
| 49. | Ram dayal | 43/M | 126/80 | None | NAD | Not done | Not done |
| 50. | Salil Bansal | 37/M | 136/74 | None | NAD | Not done | Not done |
| 51. | Smt. Noorjahan | 55yr/F | 120/80 | Burning micturition | Calcium oxalate crystals | Not done | Not done |
| 52. | Smt. Akhtari biwi | 45 yr/F | 116/72 | None | Sugar++++ | BL. Sugar 385mg/dl | Not done |
| 53. | Chand Biwi | 50/F | 118/80 | None | Protein traces 5-6 Pus cells/ HPF 2-3 epithelial cells | Not done | Not done |
| 54. | Suttan | 8/M | 100/76 | None | NAD | Not done | Not done |
| 55. | Mr.R.P.Singh | 82/M | 174/96 | None | NAD | Not done | Not done |
| 56. | Avinash | 8/M | 100/70 | None | 6-8 pm cells/HPF | Not done | Not done |
| 57. | Sumat Kumar | 40/M | 106/80 | None | NAD | Not done | Not done |
| 58. | Chandra Singh | 45 yr/M | 110/.80 | None | Proteins + | Random Blood sugar 315mg% | Not done |
| 59. | Meenu Shekhar | 15 yr/ F | 106/70 | None | NAD | Not done | Not done |
| 60. | Rajendra Prasad | 25/M | 126/80 | None | NAD | Not done | Not done |
| 61. | Sukhiya | 35/F | 110/80 | None | 4-6 per cells/HPF | NAD | Not done |
| 62. | Kamran | 26/M | 120/80 | None | Oxalate crystals seen | NAD | |
| 63. | Gajendra | 60 yr/M | 164/100 | None | Potein, ++ | Bl. Sugar(R) 126 mg% | B/L Hazy media |
| 64. | Tahir Husain | 12/M | 100/70 | Occ. Headache + Dimunition of vision | NAD | Not done | Not done |
| 65. | Vidya Devi | 28/F | 100/76 | Mild occasional Headache | NAD | Not done | Not done |
| 66. | Bhagwati Kumar singh | 40/F | 140/86 | None | NAD | Not done | Not done |
| 67. | Vivendra singh | 44/M | 132/80 | Abd dismonful | NAD | Not done | Not done |
| 68. | Jayannath singh | 42/M | 120/86 | Lt. iliac fossa pain | NAD | Not done | Not done |
| 69. | Laxmi shankar | 41/M | 120/80 | None | Traces of albumin present | Not done | Not done |
| 70. | Rampyari | 44/F | 116/72 | None | NAD | Not done | Not done |
| 71. | Ramnarayan singh | 42/M | 150/78 | Abd pain | NAD | Not done | Not done |

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|---|---|---|---|---|---|---|---|
| 72. | Vinay kumar | 16/M | 112/80 | palpitation | NAD | Not done | Not done |
| 73. | Rajna | 26/F | 104/74 | None | NAD | Not done | Not done |
| 74. | Fagruddin | 51/M | 156/80 | None | NAD | Not done | Not done |
| 75. | Kunwar behadni | 34/M | 110/70 | nasal blockade + headache | NAD | 94mg% (2hrpp) | Not done |
| 76. | Ram Kunwar | 35/m | 120/78 | None | 20-30 pus cells/ HPF 15-16 RBC/ hpf ; oxalate crystals present | 120mg% ( 3 hr pp) | Not done |
| 77. | Dhyan Tripathi | 31/M | 110/80 | Headache | NAD | Not done | Not done |
| 78. | Jashods | 40/F | 140/80 | None | NAD | Not done | Not done |
| 79.. | Panns lal | 40/M | 110/90 | None | NAD | Not done | Not done |
| 80. | Anil kumar | 28/M | 120/82 | Burning micturition for last 6-7 days | NAD | Bl. Sugar 108mg% (3hrpp) | Not done |
| 81. | Vishram singh | 30/M | 116/78 | Headache | NAD | Not done | Not done |
| 23. | Suresh pratap | 41/M | 140/80 | Epigastric pain | NAD | Not done | Not done |
| 83. | Ram kumar | 35/M | 120/74 | Cough and cold | Sugar+ | BL. Sugar = 274mg% (2hrpp) | B/L Normal |
| 84. | Ram Avbaar | 26/M | 150/104 | Headache | Protein ++ | Bl Sugar 98mg% | Papillede ms Lt. eye |
| 85. | Baboo | 6/M | Not Recorded | None | NAD | Not done | Not done |
| 86. | Prem Nath | 30/M | 126/80 | None | NAD | Not done | Not done |
| 87. | Naresh Prasad | 64/M | 105/50 | None | 6-8 per cells/HPF | Not done | Not done |
| 88. | Preetaim sinth | 50/M | 120/50 | None | NAD | Not done | Not done |
| 89. | Sukhi | 32/M | 110/70 | None | NAD | Not done | Not done |
| 90. | Amit sadan | 15/M | 120/76 | None | NAD | Not done | Not done |
| 91. | Radhika Srivastava | 23/F | 110/70 | Low grade fever | NAD | Not done | Not done |
| 92. | Anbika srivastava | 43/F | 160/100 | None | NAD | Not done | Not done |
| 93. | Kasim Iqbaal | 35/M | 120/80 | 8-10 per cells/HPF oxalate, phosphate crystals | NAD | Not done | Not done |
| 94. | Palayanutkal | 45/M | 130/50 | None | NAD | Not done | Not done |
| 95. | Mahesh pandey | 40/m | 140/50 | None | NAD | Not done | Not done |
| 96. | Gudiya | 10/F | 110/70 | None | NAD | Not done | Not done |
| 97. | Shayama | 15/F | 104/70 | Lump in breast | NAD | Not done | Refr to sugar |
| 98. | Ganesh kutums | 30/M | 120/76 | None | NAD | Not done | Not done |
| 99. | Pooran dayal | 25/M | 120/82 | None | NAD | Not done | Not done |
| 100. | Ghan shayam | 35/M | 130/76 | None | NAD | Not done | Not done |
| 101. | Rohit | 5/M | 140/70 | None | NAD | Not done | Not done |
| 102. | Peelu Bhagat | 40/M | 146/100 | obesity | Sugar + protein + | 350 mg % 3 hr pp | Not done |
| 103. | Pyaare lal | 32/M | 120/50 | None | NAD | Not done | Not done |
| 104. | Madan Kativaav | 30/M | 117/76 | None | NAD | Not done | Not done |
| 105. | Suneel jain | 25/M | 120/74 | None | NAD | Not done | Not done |
| 106. | Preeti jain | 20/F | 110/70 | None | NAD | Not done | Not done |
| 107. | Anant jain | 50/M | 110/50 | None | NAD | Not done | Not done |
| 108. | Namita Das | 23/F | 120/76 | None | NAD | Not done | Not done |
| 109. | Shobha Das | 15/F | 100/90 | Occasional burning micturition | Pus cells 10-12/HPF Protein + | Not done | Not done |
| 110. | Gaman Das | 30/M | 110/74 | None | NAD | Not done | Not done |
| 111. | Eshwar Gupta | 26/M | 130/76 | None | NAD | Not done | Not done |
| 112. | Tarun purohit | 43/M | 140/90 | Occ. Abd pain | NAD | Not done | Not done |
| 113. | Bheem kumar | 25/M | 120/70 | Scrotal pruritis | NAD | Not done | Not done |
| 114. | Sheela kumar | 22/F | 100/70 | None | NAD | Not done | Not done |
| 115. | Taranum Khan | 30/F | 140/70 | Pregnancy ( 24 weeks) asymptomatic | Pus cells 6-5/HPF | Not done | Not done |
| 116. | EhsanKhan | 36/M | 110/90 | None | NAD | Not done | Not done |

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|---|---|---|---|---|---|---|---|
| 117. | Nidhi pathak | 10/F | 100/74 | Burning micturition | Pus cells 14-16/HPF<br>Protein ++ | 97 mg %<br>( 2 hr pp) | Not done |
| 118. | Rudra Sen | 25/M | 120/70 | None | NAD | Not done | Not done |
| 119. | Rambha chaudhary | 45/F | 200/130 | None | Protein + | 294 mg%<br>( 4 hr pp ) | Not done |
| 120. | Gaya Deen | 30/M | 140/90 | None | NAD | Not done | Not done |
| 121. | Jugal | 38/M | 120/50 | None | NAD | Not done | Not done |
| 122. | Shekhar pal | 20/M | 110/76 | None | NAD | Not done | Not done |
| 123. | Neetu | 5/F | 150/90 | None | Protein++<br>Red cells = 10-14/HPF | Not done | Not done |
| 124. | Daman | 5/M | 100/74 | None | NAD | Not done | Not done |
| 125. | Ramayya | 24/M | 110/50 | Headache | NAD | Not done | Not done |
| 126. | Chandan | 26/M | 120/50 | Cough + loss of weight (1 month) | NAD | Not done | Not done |
| 127. | Kalku Prasad | 50/M | 130/86 | Chronic cough with expectoration(smoker) | NAD | Not done | Not done |
| 128. | Gomti Devi | 32/F | 100/76 | None | NAD | Not done | Not done |
| 129. | Sudha Devi | 36/F | 120/78 | ↑ bleeding during periods | NAD | Not done | Not done |
| 130. | Katori Devi | 42/F | 104/76 | None | NAD | Not done | Not done |
| 131. | Digam babu | 33/M | 130/82 | None | NAD | Not done | Not done |
| 132. | Alatas Khan | 40/M | 145/94" | Obesity | Sugar + +<br>Pus cells 8-10/HPF | 294mg%<br>(3 hr pp) | Not done |
| 133. | Kumari Suneeta | 17/F | 110/70 | None | NAD | Not done | Not done |
| 134. | Jeevan lal | 25/M | 140/100 | None | NAD | Not done | Not done |
| 135. | Gaurav lal | 30/M | 160/60 | None | NAD | Not done | Not done |
| 136. | Prem bai | 44/F | 120/68 | None | NAD | Not done | Not done |
| 137. | Satm Singh | 37/M | 130/90 | None | NAD | Not done | Not done |
| 138. | Sumi Shukla | 26/F | | None | NAD | Not done | Not done |
| 139. | Prakash Shukla | 30/M | | None | Phosphate & urate crystals<br>+<br>RBC = 10-15/hpf | Not done | Not done |
| 140. | Chanchal shukla | 5/F | 140/100 | None | Protien ++ | 78 mg %<br>( 4 hr pp ) | Not done |
| 141. | Sagun bai | 30/F | | None | NAD | Not done | Not done |
| 142. | Triveni sood | 32/F | | None | NAD | Not done | Not done |
| 143. | Arjun Singh | 20/M | | None | Pus cells 10-15 /HPF | 121 mg %<br>( 3 hr pp) | Not done |
| 144 | Nakul singh | 35/M | | None | NAD | Not done | Not done |
| 145. | Shobha Ram | 48/M | 120/50 | Fever 2-3 days with cough | NAD | Not done | Not done |
| 146. | Tasar chaube | 27/M | 126/70 | None | NAD | Not done | Not done |
| 147 | Shivani Shukls | 32/F | 136/94 | None | NAD | Not done | Not done |
| 148. | Brij kishore Rai | 35/M | 160/110 | None | Protien++ | Bl. Gluc 92mg% 4hrpp | Diadetic retinopathy |
| 149. | Umar Mukhtar | 42/M | 144/94 | None | NAD ( only occasioned pus cells seen) | Bl. Glucose 254mg% | Diabetes Retiniply |
| 150. | Pyaare Mohan | 32/M | 124/76 | None | Oxalate and phosphate crystals | Not done | Not done |
| 151. | Dilip Dhavia | 28/M | 130/90 | None | NAD | Not done | Not done |
| 152. | Upendra Baijal | 32/M | 120/50 | None | NAD | Not done | Not done |
| 153. | Kahar singh | 24/M | 190/110 | None | Protein=+ | 140 mg %<br>( 2 hr pp) | B/L WNL |
| 154. | Doli Mehta | 16/F | 105/70 | Itching all over body | NAD | Not done | Not done |
| 155. | Lagan Gupta | 58/M | 149/90 | Poor stream formation during urination | Pus cells 10-12/HPF | 136mg%<br>( 3 ½ hr pp) | Not done |
| 156. | Anand puri | 64/M | 150/100 | None | NAD | Not done | Not done |
| 157. | Shamshad Ansari | 45/M | 120/80 | Itching around the anus | NAD | Not done | Not done |
| 158. | Anwari begum | 35/F | 116/70 | Weekness+ pain in limbs | NAD | Not done | Not done |

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|---|---|---|---|---|---|---|---|
| 159. | Pista Devi | 40/F | 100/70 | None | NAD | Not done | Not done |
| 160. | Prabhu bhagat | 16/M | 118/80 | None | NAD | Not done | Not done |
| 161. | Peelu Singh | 44/m | 134/80 | None | Protein =+++ | Not done | Not done |
| 162. | Deewakar purohit | 50/M | 120/50 | None | None | Not done | Not done |
| 163. | Mandeep chadha | 45/m | 150/100 | Burning micturition | Pus cells 20-25/HPF Protein ++ | 314mg% | Diabetic retinopat y seen |
| 164. | Pramesh Tiwari | 30?M | 124/78 | None | NAD | Not done | Not done |
| 165. | Shivangi Misra | 34/F | 110/70 | None | NAD | Not done | Not done |
| 166. | Bahusali | 40/F | 138/74 | None | NAD | Not done | Not done |
| 167. | Preetam Srivastava | 22/M | 198/120 | None | Protein ++ | 100 mg % ( F) | B/L Vessel changes no papillede ma. |
| 168. | GauravAtareja | 30/M | 126/82 | None | NAD | Not done | Not done |
| 169. | Rajan Gupta | 32/M | 110/70 | None | NAD | Not done | Not done |
| 170. | Sugoto Mukherjee | 25/M | 126/84 | None | NAD | Not done | Not done |
| 171. | Deewan Seth | 58/M | 160/100 | None | Protein=++ Pus cells =10-12/HPF | 100mg% | could not be seen B/L cataract |
| 172. | Prem Das | 70/M | 140/96 | Known diabetic on drugs | Protein=++ | 212mg% | B/L cataract |
| 173. | Nimal Saxena | 34/M | 126/70 | None | NAD | Not done | Not done |
| 174. | Gopal Agarwal | 15/M | 110/70 | None | NAD | Not done | Not done |
| 175. | Bheemsen | 34/M | 124/88 | None | Plenty of calcium & oxalate crystals | Not done | Not done |
| 176. | Gonda Ram | 12/m | 104/100 | None | None | Not done | Not done |
| 177. | Madhuri Gupta | 5/F | 110/70 | None | Sugar +++ | 250mg% (2 hr pp) | B/l Normal |
| 178. | Nitesh Kumar | 5/M | 100/70 | None | NAD | Not done | Not done |
| 179. | Sanwal Pavihaar | 35/M | 174/100 | None | NAD | Not done | Not done |
| 180. | Kastoori Devi | 23/F | 110/76 | None | NAD | Not done | Not done |
| 181. | Urmila sethi | 44/F | 130/84 | None | Sugar ++ Pus cells 15-20/hpf | 340mg% (2hrpp) | Not done |
| 182. | Videshi prakash | 70/M | 120/84 | None | NAD | Not done | Not done |
| 183. | Nimnum Shukla | 80/M | 160/90 | None | NAD | 138mg% (3hrpp) | Not done |
| 184. | Susheel chaturvedi | 24/m | 120/78 | None | NAD | Not done | Not done |
| 185. | Uttam gorda | 60/M | 120/50 | None | NAD | Not done | Not done |
| 186. | Surrabh singh | 74/M | 140/50 | None | NAD | Bl. Sugar 150mg% | Not done |
| 187. | Mahesh pandey | 25/M | 126/90 | None | NAD | Not done | Not done |
| 188. | Sachin Agarwal | 50/M | 130/70 | None | NAD | Not done | Not done |
| 189. | Pardhan Kumar | 74/m | 130/94 | None | NAD | 94mg% (1hrpp) | Hyperten sive vessel changes |
| 190. | Mithu | 6/F | 154/100 | None | NAD | 100mg%(R) | B/L normal |
| 191. | Gauri Devi | 30/F | 110/70 | None | NAD | Not done | Not done |
| 192. | Susheela Devi | 50/F | 100/76 | None | Pus cells 12-14/HPF | 112mg%(R) | None |
| 193. | Samreth Gupta | 20/M | 120/56 | None | NAD | Not done | Not done |
| 194. | Nisbukl singh | 16/M | 126/50 | None | NAD | Not done | Not done |
| 195. | Azam beg | 42/M | 10/50 | None | NAD | Not done | Not done |
| 196. | Kamini Begum | 38/F | 150/100 | None | Protein=++ | 349mg%(R) | Diabetic Retinopat hy |
| 197. | Ayaz beg | 14/M | 120/50 | None | NAD | Not done | Not done |
| 198. | Lomvati | 25F | 105/70 | None | NAD | Not done | Not done |

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|---|---|---|---|---|---|---|---|
| 199. | Sadhna sethi | 24/F | 110/50 | None | NAD | Not done | Not done |
| 200. | Ramayya Devi | 30/F | 104/70 | Burning micturition | Pus cells 10-12/HPF<br>Protein + | 100mg%(R) | Not done |
| 201. | Samved misra | 24/M | 124/80 | None | NAD | Not done | Not done |
| 202. | Rajini misra | 60/F | 150/100 | None | NAD | 94 mg % | Not done |
| 203 | Ayur kumar | 56/M | 130/84 | None | NAD | Not done | Not done |
| 204 | Malkhan | 44/M | 140/96 | None | NAD | Not done | Not done |
| 205 | Ninhai kumar | 32/M | 140/80 | None | NAD | Not done | Not done |
| 206. | Dhaniram | 26/M | 114/50 | None | NAD | Not done | Not done |
| 207. | Vishwanath Das | 54/M | 110/70 | Poor Stream formation | Protein (+) | 109mg%(R) | Not done |
| 208. | Gajendra singh | 64/M | 110/70 | None | NAD | Not done | Not done |
| 209. | Chatur singh | 20/M | 108/76 | None | NAD | Not done | Not done |
| 210. | Meera Devi | 68/F | 120/74 | None | Sugar ++<br>Protein ++<br>Pus cells 10-12/HPF | 274mg%(R) | B/L Cataract |
| 211. | Babu lal | 34/M | 110/70 | None | NAD | Not done | Not done |
| 212. | Magbool Khan | 36/M | 124/50 | None | NAD | Not done | Not done |
| 213. | Mashkool beg | 72/M | 150/100 | Poor urinary stream | NAD | 108mg%<br>(2 hr pp) | Not done |
| 214. | Sajid Husain | 9/M | 160/100 | Swelling around eyes | Protein ++ | 97mg%<br>(3 hr pp) | hypertensive changes |
| 215. | Dharmendra Kumar | 12/M | 150/98 | None | NAD | Not done | Not done |
| 216. | Sheela Devi | 22/F | 100/70 | Occasional burning micturition | NAD | Not done | Not done |
| 217. | Ladkunwar | 65/F | 110/70 | Burning micturition + prolapse | Protein +<br>Pus Cells 14-16/hpf | 112mg%(R) | Not done |
| 218. | Mankunwar | 58/F | 210/120 | Occasional headaches | Protein=++ | 114 mg %<br>(R) | B/L Cataract |
| 219. | Altaaf Raza | 36/m | 124/50 | None | NAD | Not done | Not done |
| 220. | Gaman Tyagi | 16/M | 120/50 | None | NAD | Not done | Not done |
| 221. | Sumit sood | 22/M | 110/74 | None | NAD | Not done | Not done |
| 222. | Pyaari | 24/F | 104/70 | None | Pus cells 8-10/HPF | 94mg% | Not done |
| 223. | Gurjani Devi | 42/F | 110/70 | None | Pus cells 16-18/HPF | 174mg%<br>(F) | Not done |
| 224. | SringarDevi | 30/F | 132/50 | None | NAD | Not done | Not done |
| 225. | Gita Devi | 16/F | 110/76 | None | NAD | Not done | Not done |
| 226. | Naseeb ullah. | 39/M | 120/84 | None | NAD | Not done | Not done |
| 227. | Suman | 5/F | Not.Rec. | None | NAD | Not done | Not done |
| 228. | Dhaniram | 50/M | 120/54 | None | NAD | Not done | Not done |
| 229. | Indu Gupta | 16/F | 110/70 | None | NAD | Not done | Not done |
| 230. | Ramdas | 74/M | 150/104 | None | NAD | 84mg% | B/L Cataract |
| 231. | Sanjay Tiwari | 24/M | 120/50 | None | NAD | Not done | Not done |
| 232. | Kamla Tiwari | 20/F | 104/70 | None | NAD | Not done | Not done |
| 233. | TriveriGupta | 5/F | 140/90 | None | NAD | 110mg%(R) | Fundus Normal |
| 234. | Naman Gupta | 34/M | 120/50 | None | NAD | Not done | Not done |
| 235. | Hardas Singh | 727M | 110/50 | Poor stram of urine+taken long time to pass urine | NAD | BL Sugar 124mg%(R) | B/L Cataract |
| 236. | Guddi | 6/F | 100/70 | None | NAD | Not done | Not done |
| 237. | Purnima | 5/F | 110/80 | None | 10-12 pus cells/HPF | 154mg%(R) | |
| 238. | Bhagwan das | 47/M | 1201/56 | None | NAD | Not done | Not done |
| 239. | Baby singh | 2/F | No recorded | None | NAD | Not done | Not done |
| 240. | Malkan singh | 30/M | 120/82 | None | 10-12 Pus cells/HPF | 132mg%(R) | |
| 241 | Sevaknath | 14/M | 120/54 | None | NAD | Not done | Not done |
| 242. | Dasnishkamal | 25/M | 124/76 | None | NAD | Not done | Not done |

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|---|---|---|---|---|---|---|---|
| 243. | Premvati | 32/F | 110/70 | Occ. Renal angle pain | Pus cells 6-8/HPF oxalate + phosphate crystals | Not done | Not done |
| 244. | Deewan singh | 40/M | 110/50 | None | NAD | Not done | Not done |
| 245. | RaniDevi | 33/F | 120/50 | None | NAD | Not done | Not done |
| 246. | Rouma singh | 16/F | 100/72 | None | 10-12Pus cells/HPF | Not done | Not done |
| 247. | Subedarsingh | 436/M | 120/5/8 | None | NAD | Not done | Not done |
| 248. | Kiran kumari | 5/F | 110/70 | None | NAD | Not done | Not done |
| 249. | Ganga Devi | 66/F | 178/98 | None | NAD | 96mg%(R) | B/L Hazy media |
| 250. | Om Dev | 59/M | 150/54 | Known diabetic (on drugs) | Protein + | Bl. Sugar =166 | B/L Cataract |
| 251. | Sripal singh | 45/M | 120/90 | None | NAD | Not done | Not done |
| 252. | Allen Dsouza | 55/M | 188/104 | None | Pus cells 6-8/hpf protein + | 104mg%(R) | Hyprtensive vessel changes |
| 253. | Kareem | 16/M | 110/80 | None | NAD | Not done | Not done |
| 254. | Barkat | 45/M | 110/54 | None | Pus cells=10-12/HPF | 160mg%(R) | Not done |
| 255. | Laadli | 8/F | 120/80 | None | NAD | 75mg% (5hrpp) | Not done |
| 256. | Aroop kundu | 35/M | 126/70 | None | NAD | Not done | Not done |
| 257. | Sadhna kumari | 3/F | Not recorded | None | NAD | 97mg%(R) | Not done |
| 258. | Bilqees Bano | 32/F | 110/70 | None | NAD | Not done | Not done |
| 259. | Shanaaz Bano | 16/F | 100/70 | None | NAD | Not done | Not done |
| 260. | Rahman Ullah | 50/M | 150/96 | Inabiliy to fall asleep | Pus cells=8-10/HPF | Bl. Sugar =84mg%(R) | B/L Cataract |
| 261. | Teerath Nath | 49/M | 130/70 | None | NAD | Not done | Not done |
| 262. | Baba | 24/M | 120/74 | None | NAD | Not done | Not done |
| 263. | Kalloo | 33/M | 116/70 | None | NAD | Not done | Not done |
| 264. | Sagarika Das | 23/F | 100/70 | Burning micturition | Pus cells 16-20/HPF | 88mg% (2hrpp) | Not done |
| 265. | T. Mahapatra | 25/M | 110/50 | None | NAD | Not done | Not done |
| 266. | Amrit Singh | 34/M | | None | NAD | Not done | Not done |
| 267. | Leena Devi | 40/f | 105/70 | None | NAD | Not done | Not done |
| 268. | PrabhuDayal | 45/M | 130/86 | None | NAD | Not done | Not done |
| 269. | Gayatri Devi | 62/F | 140/50 | Known diabetes on drugs | NAD | 210mg%(2hrpp) | B/L Cataract |
| 270. | Eeshwar Das | 59/M | 170/90 | None | NAD | Bl. Sugar=84mg% (R) | B/L( N) |
| 271. | Ram Khilawan | 41/M | 110/70 | Occasional renal angle pain | Pus cells 8-10/hpf Oxalate crystals + | Not done | Not done |
| 272. | Krishna Sengar | 32/F | 100/70 | None | NAD | Not done | Not done |
| 273. | Gautam Jain | 47/M | 110/70 | None | NAD | Not done | Not done |
| 274. | Govind Singh | 54/M | 120/70 | None | NAD | 94mg% (2hrpp) | (N) |
| 275. | Meena | 5/F | 110/70 | None | NAD | 75g%(4hrpp) | B/L (N) |
| 276. | Veena | 26/F | 104/70 | None | NAD | Not done | Not done |
| 277 | Katori Devi | 33/F | 1400/68 | None | 12-15/HPF Pus cells | Bl. Sugar=72mg% | Not done |
| 278. | Kalyan | 35/M | 120/78 | None | NAD | Not done | Not done |
| 279. | Gaman singh | 24/M | 110/70 | None | NAD | Not done | Not done |

# PART II –FOLLOW UP OF PATIENTS WITH ASYMPTOMATIC URINARY ABNORMALITIES

| S.No | Name | Age/ Sex | Blood Pressure in mmHg | Symptoms | S. Creatinine | Repeat Urine (R,M) | USG for KUB | Biopsy/ Culture | Blood Glucose Fasting | Diagnosis |
|---|---|---|---|---|---|---|---|---|---|---|
| 1 | Shanti Bai | 55/F | 140/96 | H/O swelling of whole body since last 2 years and abdominal pain 1 month | 6.7 | Protein++ | B/L Shrunken kidney | Not done | 94mg% | Symptomatic CRF |
| 2 | Smt. Sushma shivhara | 32/F | 120/72 | Burning micturition last 3 days | 0.9 | 10-12 pus cells\HPF | Normal kidney size with normal corticome dullary images I | E.coli in culture | 77mg% | UTI |
| 3. | Ram lal | 64/M | 130/90 | Asymptomatic | 0.7 | Sugar++ puscells-6-8/HPF | NAD | Not done | 321mg% | Diabetes Mellitus with U.T.I. |
| 4. | Anjana | 36/F | 120/80 | Asymptomatic | 0.8 | Sugar+++ puscells14-16/HPF | NAD | Not done | 194mg% | Diabetes Mellitus |
| 5. | Mohd Mustaq | 51/M | 150/98 | Polydipsia polyurea | 1.1 | Sugar+++ puscells 4-6/HPF | NAD | Not done | 248mg% | Diabetes Mellitus |
| 6. | Kirti | 10/F | 110/74 | Asymptomatic | 1.2 | Protein++ | B/L Increased Cortical Echogenicity | Biopsy shows Focal segmental glomerulosclerosis | 98mg% | focal segmental glomerulosclerosis |
| 7. | L.K. Sharma | 46/M | 124/80 | Asymptomatic | 1.1 | Puscells 10-15/HPFOxalate crystals++Phosphats+ | B/L Renal Calculus | Not done | | B/L nephrolithiasis |
| 8. | Bhagwati | 40/F | 140/94 | Arthralgia+insomnia | 1.0 | 10-12 Pus cells/HPF | NAD | Not done | 234mg% | Diabetes Mellitus |
| 9. | Hadid khan | 65/M | 130/84 | Asymptomatic | 1.4 | Protein++ Puscells6-8/HPF | B/Lmild Hydronephrosis with grade II BPH | Not done | 50mg% | B.p.H. with U.T.I. |
| 10- | Mohd Mustaq | 51/M | 120/82 | Pain at the Tip of penis during micturition | 0.5 | Field full of pus cells | 25mm renal calculus in the renal pelvis | Not done | 84mg% | nephrolithiasis |
| 11. | Raj Kumari | 35/F | 130/84 | Known C/O type II DM ( M drugs) | 1.0 | Protein+ | NAD | Note done | 165mg% | Known Diabetic |
| 12. | Noorjahan | 55/F | 120/76 | Micturition | 0.7 | Oxalate Crystals ++ | B/L multiple renal calculli | Not done | 90mg% | B/L nephrolithiasis |
| 13. | Akhtari bibi | 45/F | 130/80 | Asymptomatic | 1.0 | Sugar++ | NAD | Not done | 245mg% | Diabetes Mellitus |

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|---|---|---|---|---|---|---|---|---|---|---|
| 14. | Chand bibi | 50/F | 120/70 | Asymptomatic | 1.19 | Pus cells 8-10/HPF | NAD | | 94mg% Culture Shows E. coli growth | U.T.I. |
| 15. | Avinash | 8/M | 110/70 | Asymptomatic | 1.14 | Pus cells6-8/HPF | NAD | Voiding pyelogram shows reflux | 100mg% | Vescico ureteral reflux |
| 16 | Chandra singh | 45/M | 124/80 | Asymptomatic | 0.9 | Protein+ | NAD | Not done | 225mg% | Diabetes Mellitus |
| 17. | Sukhiya | 35F | 110/74 | Asymptomatic | 0.97 | 8-10Pus cells/HPF | NAD | E.coli growth in culture | 80mg% | U.T.I. |
| 18. | Kamran | 26/M | 110/70 | Asymptomatic | 0.84 | Abundant calcium oxalate cmprtal1-2 pus cells/HPF | 7.5mmCalculus in lt Kidney | Not done | 112mg% | Nephrolithiasis |
| 19. | Gajendra | 60/M | 174/110 | Asymptomatic | 1.1 | Protein++ | NAD | Biopsy shows Bening Nephrotic disease | 104mg% | Hypertensive Nephropathy |
| 20. | Laxmi Shanker | 41/M | 200/120 | Asymptomatic | 0.74 | Protein+ | NAD | Benign Nephrosclorosis | 95mg% | Hypertensive Nephropathy |
| 21. | Ramkunvar | 38/M | 120/74 | Asymptomatic | 0.91 | Pus cells16-18/HPF oxalatecryted plsent | B/LRenal Calculus | Not done | 174mg% | Diabetes Mellitus |
| 22. | Ramkumar | 35/M | 146/98 | Cough+Lold | 0.72 | Sugar++occasioned pus cells | NAD | Not done | 174mg% | Diabetes Mellitus |
| 23. | Ram Avtaar | 26/M | 150/106 | Occasional headache | 0.97 | Potein++ | Grade II Renal Parenchyncl disease | Bipsy showsCGN | 96mg% | CGN |
| 24. | Nareshprasad | 64/M | 140/100 | Asymptomatic | 1.0 | Pus cells8-10/HPF | Grade I BPH | Not done | 104mg% | B.P.H |
| 25. | Kasim Iqbaal | 35/M | 120/80 | Asymptomatic | 1.1 | Pus cells 6-8/HPF RBCs 5-7/HPF Oxalate + phosphate crytals | Renal calculus Rt Kidney | Not done | 74mg% | Nephrolithiasis |
| 26. | Peelu Bhagat | 40/M | 126/74 | Obesity | 0.7 | Sugar++ Protein+ | NAD | Not done | 251mg% | Diabetes Mellitus |
| 27. | Shobha Das | 15/F | 110/74 | Occasional buriney micturition | 0.8 | 10-12 Pus cells /HPF | NAD | Culture shows E coli | 100mg% | U.T.I. |
| 28. | Tayanum Khan | 30/F | 100/70 | Asymptomatic | 0.78 | Pus cells 10-12/HPF | Normal foctus of 26 week gst age KUB Normal | Not done | 110mg% | U.T.I. |
| 29. | Nidhi Pathak | 10/F | 110/90 | Burning micturition | 0.79 | Pus cells10-14/HPF | NAD | Culture shows klebsiella | 84mg% | U.T.I. |
| 30. | Rambha Chaudhary | 45/F | 204/120 | Asymptomatic | 0.67 | Protien++ | NAD | Biopsy shows benign nephro sclerosis | 110mg% | Hypertensive nephropathy |

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|---|---|---|---|---|---|---|---|---|---|---|
| 31 | Neetu | 8/F | 150/90 | Asymptomatic | 1.12 | Protein ++ RBC + | NAD | | 84mg% | IGA Nephropathy |
| 32. | Alatash Khan | 40/M | 148/150 | Asymptomstic | 0.87 | Sugar ++ Protein + Puscells 8-10/HPF | NAD | Not done | 221mg% | Diabetic Mellitus |
| 33. | Prakash Shukla | 5/F | 1401/100 | Asymptomatic | 1.8 | Protein++ | B/L renal parenchyn al disease Rt kidney size =0.57 x 4.3 mmRt kidney size normal | Not done | 73mg% | Nephrolithiasis |
| 34 | Arjun Singh | 20/M | 124/76 | Asymptomatic | 0.8 | Pus Cells 10-15/HPP | NAD | Streptococci growth | | U.T.I. |
| 35 | B. K. Rai | 35/M | 160/116 | Asymptomatic | 0.94 | Protein ++ | NAD | Biopsy shows Benign Nephrosis | 80mg% | Hypertensive Nephropathy |
| 36. | Umar Mukhtar | 42/M | 150/100 mmHg | Asymptomatic | 0.83 | Pus cells10-12 /HPF | NAD | Not done | 270mg% | Diabetes Mellitus with U.T.I. |
| 37. | Pyaare mohan | 32/M | 124/76 | Asymptomatic | 01.71 | Oxalate +phorpha te crystals | 27 mmRt renal calculus | Not done | 120mg% | Nephrolithiasis |
| 38. | Kuhar Singh | 24/M | 200/100 | Asymptomatic | 0.98 | Protein + | USG shows increased echogenici ty | Biopsy shows feature of CGN | 80 mg% | Chronic GlomeruloNephriti s |
| 39. | Lagan gupta | 58/M | 140/86 | Poor steam formation during micturition | 0.92 | Pus cells 16-18/HPF | Grade II BPH others-NAD | Not done | 104mg% | B.P.H. |
| 40 | Peelu singh | 44/M | 128/80 | Asymptomatic | 0.94 | Protein++ | B/L grade II renal parenchy mal disease | Biopsy shows Amyloid Deposition | 88mg% | Renal Amyloidosis |
| 41. | Man deep chaddha | 45/M | 160/104 | Burning micturition | 1.2 | Pus cells full protein ++ | B/L Increased Echogenici ty | Not done | 294mg% | Diabetes Mellitus |
| 42. | Preetam Srivasta va | 22/M | 198/120 | Asymptomatic | 1.02 | Protein++ | B/L renal parenchy mal disease (gradeII) | Biopsy shows CGN | 93mg% | Chronic Glomerulo Nephritis |
| 43. | Deewan Seth | 58/M | 160/100 | Asymptomatic | 1.8 | Protein + Pus cells 6-8/HPF | B/L Renal parenchy mal disease grade I + BPH grade II | Biopsy shows Benign glomerulo sclerosis | 81mg% | BPH with Hypertensive Nephropathy |
| 44. | Prem Das | 70/M | 148/98 | Know diabetes drugs | 1.0 | Protein ++ | NAD | Not done | 174mg% | Diabetes Mellitus |

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|---|---|---|---|---|---|---|---|---|---|---|
| 45. | Bheem Sen | 34/M | 120/80 | Asymptomatic | 0.94 | crystas oxalate enypets | NAD | Biopsy Normal, serum calcium 16mg%. | 94mg% | hyperparathyroidism |
| 46. | Madhuri Gupta | 8/F | 110/70 | Asymptomatic | 0.84 | Sugar ++ | NAD | Not done | 108mg | fanconi's syndrome |
| 47. | Urmila Sethi | 44/F | 130/50 | Asymptomatic | 0.73 | Sugar +++ | 8mm size calculus in the Rt lower ureter with 5mm calculus in the Rt upper caly | Not done | 204mg% | Diabetes Mellitus with Renal Calculus disease |
| 48. | Pardhan kumar | 74/M | 174/110 | Asymptomatic | 0.81 | Protein++ | Grate II renal yarnechyal disease | Biopsy shows benign Nephro sclerosis | 93mg% | Hypertensive Renal Disease |
| 49. | Susheela Devi | 50/F | 100/70 | Asymptomatic | 0.79 | Puscells1 4-16/HPF | NAD | Culture shows E. cells | 100mg% | U.T.I. |
| 50. | Kamini Begum | 35/F | 150/100 | Poor vision | 1.20 | Protein ++ | NAD | Not done | 249mg % | Diabetes Mellitus |
| 51. | Ramayya Devi | 30/F | 104/70 | Burning micturition | 0.95 | Pus cells 14-16/HPF With traces of Albumin | NAD | Culture chows E-coli | 89mg% | U.T. I |
| 52. | Vishwanath Das | 54/M | 140/100 | Asymptomatic | 0.8 | Protein+ | USG shows grade II BPH | Not done | 77mg% | BPH |
| 53. | Meera Devi | 65/F | 120/50 | Asymptomatic | 1.24 | Sugar +++ Protein + | NAD | Not done | 344mg% | Diabetes Mellitus |
| 54. | Sajid hussain | 9/M | 160/104 | Puffiness of face & around eyes | 1.64 | Protein ++ | B/L Grade II Renal parenchymal disease | Biopsy shows C.G. N. | 94 mg% | C.G.N. |
| 55. | Ladhun war | 65/F | 110/70 | Burning during micturitum + proloper of uterim | 1.12 | Pus cells 10-12/HPF Traces of Albumin | NAD | Urine culture shows klebsiella | 74mg% | U.T.I. with prolapse uterus |
| 56. | Maan kunwar | 58/F | 210/120 | Asymptomatic | 1.69 | Protein ++ | NAD | Biopsy show Benign nephrosclerosis | 70mg% | Hypertensive Nephropathy |
| 57. | Pyaari | 24/F | 100/70 | Asymptomatic | 0.77 | Pus cells 6-10/HPF | NAD | Not done | 82mg% | U.T.I. |
| 58. | Ganjani Devi | 42/M | 110/76 | Asymptomatic | 0.72 | Pus cells 16-18/HPF | Normal | Not done | 170mg% | DiabeteS Mellitus |
| 59. | Malkhan singh | 30/M | 120/50 | Asymptomatic | 0.91 | 12-14 Pus cells/HPF | B/L renal calculus in the upper calyces(Rt. 8mm;Rt=12mm) | Not done | 75mg% | Nephrolithiasis |

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|---|---|---|---|---|---|---|---|---|---|---|
| 60. | Premvat i | 32/F | 110/70 | B/L occ. Renal angle pain | 0.84 | Pus cells 10-12/HPF few phosphat e cryplidd with oxalate cryrtas | B/L renal calcular disease (Rt 24mm cacular at low calyers ) (Rt 10 mm calcular ain the Pelvo ) | Not done | 72mg% | Nephrolithiasis |
| 61. | Rooma singh | 16/F | 110/72 | Asymptomatic | 0.77 | 14-16 Pus cells/HPF | NAD | Culture shows E-coli | 94mg% | U.T.I. |
| 62. | Om Dev | 59/M | 150/94 | Known diabetes on drugs | 1.0 | Protein ++ | NAD | Not done | 200mg% | Diabetes Mellitus |
| 63. | Allen Dsouza | 35/M | 188/104 | asymptomatic | 2.0 | Protein + RBCs 6-8/HF | NAD | Biopsy shows Benign nephso | 92mg% | Hypertensive Renal disease |
| 64. | Barkat | 45/M | 110/80 | asymptomatic | 0.72 | Pus cells 10-12/HPF | NAD | Culture shows klebsiells | 104mg% | U.T.I. |
| 65. | Rahman wllak | 50/M | 160/100 | Asymptomatic | 0.92 | 8-10Pus cells /HPF RBC 6-8/HPF | Grate II BPH | Biopsy – Normal | 89mg% | BPH |
| 66. | Saganik a Das | 23/F | 110/70 | Burning micturition | 0.83 | Pus cells 6-8/HPF | NAD | E.Coli in culture | 50mg% | UTI |
| 67. | Ram khilawa n | 41/M | 116/70 | Rt Renal angle pain( occasimal ) | 1.20 | Pus cells 8-10/HPF Occasinal oxalate crystals seen | A small 8mm calculus seen at the Rt lower calyx of the Rt gidney | Not done | 74mg% | Nephrolithiasis |
| 68. | Katori Devi | 33/F | 100/70 | Asymptomatic | 0.7 | 16-18 Pus cells /HPF | normal | E.Coli in culture | 72mg% | UTI |